Complex Shoulder Instability: A combined study of functional MRI, Electromyography and 3-D motion analysis.

Thesis submitted in accordance with the requirements of the University of Liverpool for the degree of Doctor in Philosophy by Anthony John Howard

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Abstract : Complex Shoulder Instability : A combined study of functional MRI, Electromyography and 3-D motion analysis. - Anthony Howard

Purpose of Study : The pathophysiology of type II/III shoulder instability under the Stanmore Classification is not understood. This absence of knowledge prevents treatment strategies being devised or a proper understanding of existing therapies. This is the first study to approach this group of patients from both a cerebral and muscle analysis perspective.

Methods : The assessment of shoulder movement was undertaken using two simple movements, forward flexion and abduction. The muscles around the shoulder (AD, MD, PD, UT, SA, BB, TM, LD, PM, SSP, ISP and SUB) were assessed in 21 individuals in the standing and supine position using EMG. In the supine position the movement was restricted to the movement possible in a Siemens 1.5 Tesla MRI Scanner. Patients were recruited with Polar type II/III shoulder instability, with their inclusion confirmed by the senior surgeon and physiotherapist. In total, 16 Polar type II/III patients were recruited along with 16 age-matched controls. The patients and the controls underwent an fMRI and EMG. The fMRI protocol involved movements of forward flexion and abduction in a 1.5 tesla MRI Scanner. The EMG movements tested were forward flexion and abductions to 90 degrees (AD, MD, PD, UT, SA, TM, LD, PM, BB, ISP). Both the patients and the controls completed questionnaires: the Western Ontario Shoulder Instability Index (WOSI), Oxford Shoulder Instability Score (OSIS) and Beck's Depression Inventory.

Results : Analysis of the EMG data in the normal shoulder group confirmed activations in both supine and standing positions; however the activations in the supine position were of a different character. There was increased activation in the patient group compared to the control group. In the patient group, with a voxel level familywise error rate (FWER) $p=0.04$, there was a unique activation at MNI coordinates -38 -26 56. The cluster FWER $p<0.001$ showed additional clusters in the patient group in the Primary somatosensory cortex, BA 3, Primary Motor Cortex, BA 4, Premotor cortex, BA 6 and Dorsolateral prefrontal cortex, BA 9. When the WOSI and OSIS were used as a contrast, activations were seen in primary somatosensory cortex, BA 3, supplementary motor cortex, BA 11, orbitofrontal area, BA 26, cingulate gyrus and the amygdala. The WOSI and OSIS showed a dramatically different score in the patient group compared to the controls, save for one patient whose symptoms had largely resolved following muscle patterning physiotherapy.

Conclusion : The EMG studies in the standing and the supine position confirmed the validity of the fMRI paradigm. The instability questionnaires, WOSI and OSIS confirmed the patient group selection. The unique activation (MNI -38 -26 56) occurred within the primary motor cortex, with the cluster level voxels stretching between both the somatosensory cortex and the motor cortex. The WOSI and OSI comparisons show similar activations. This is thought to be evidence of compensatory activation. This additional activation was also seen in the EMG analysis, with evidence throughout all of the muscles that greater activation was needed to complete simple movement. Overall, the comparative addition cortical and muscles activations in patient group simultaneously demonstrate dysfunction and compensatory strategies employed to achieve simple shoulder movement.

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Personal Dedication

I would like to personally dedicate this work to my brother Stephen.

Through six degrees, numerous pieces of legal work and personal correspondence, he has with patience managed to find time in his busy schedule to proofread my work. His gentle manner in dealing with this onerous task is truly breath taking.

With my stupid dyslexic head, none of my academic achievements would have been possible without his help and I am truly blessed to have such a great brother in my life.

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Presentations

International - Podium

- Is shoulder instability all in the mind? A Howard, J Powell, D Hawkes, J Gibson, O Alizadehkhaiyat, M Roebuck, G Kemp S Frostick, 13th International Congress of Shoulder and Elbow Surgery, Korea, 18th – 20th May 2016.

- An electromyography study of muscle patterning in patients with complex shoulder instability, A Howard, D Hawkes, O Alizadehkhaiyat,, G Kemp, S Frostick, International Society of Electrophysiology and Kinesiology, Rome, July 15-18, 2014,

- Stratifying shoulder pathology: a novel application of the movement deviation profile, D Hawkes, A Howard, O Alizadehkhaiyat, A Fisher, D Groves, C Denby, G Kemp, K McIver; J Richards, S Frostick, International Society of Electrophysiology and Kinesiology, Rome, July 15-18, 2014,

- What functional magnetic resonance imaging (fMRI) tells us about Complex Shoulder Instability, A Howard, D Hawkes, J Gibson, O Alizadehkhaiyat, M Roebuck, G Kemp S Frostick, American Academy of Orthopaedic Surgeons Conference,11-New Orleans, LA, March 15, 2014,.

- Simultaneous functional magnetic resonance imaging (fMRI), electromyography (EMG) and motion capture (MC) of the shoulder, A Howard, D Hawkes, M Robinson, J Gibson, K MacIver, O Alizadehkhaiyat, M Roebuck, A Fisher, G Kemp, Gabor Barton, Simon Frostick, International Congress of Shoulder and Elbow Surgery, Nagoya, Japan, April 10-12, 2013.

International – Poster

- Are the brains of patients with complex shoulder instability wired differently? A Howard, J Powell, D Hawkes, J Gibson, O Alizadehkhaiyat, M Roebuck, G Kemp S Frostick, American Academy of Orthopaedic Research Society, Las Vegas, NV, 28 March- 1 April, 2015. – JUDGED BEST POSTER IN SHOULDER/ELBOW and invited to present at AAOS 2015.

- Is the neural control different in complex shoulder instability patients? A Howard, J Powell, D Hawkes, J Gibson, O Alizadehkhaiyat, M Roebuck, G Kemp S Frostick, American Academy of Orthopaedic Research Society, Las Vegas, NV, 28 March- 1 April, 2015,

- Correlation of the Movement Deviation Profile of shoulder muscle EMG with measures of shoulder function, G Barton, D Hawkes, O Alizadehkhaiyat, A Howard, M Roebuck, A Fisher, S Frostick, M Robinson, M Hawken, International Society of Electrophysiology and Kinesiology, July 12-21, 2012, Brisbane, Australia (accepted but not presented);

- An electromyography analysis and motion analysis of the range of movement within a MRI scanner, A Howard, D Hawkes, O Alizadehkhaiyat, A Fisher, G Kemp, B Bimson, D Groves, J Richards, S Frostick, ORS Annual Meeting in San Francisco, California, February 2012;

European – Podium

- Cortical activation in patients with complex shoulder instability, A Howard, D Hawkes, J Gibson, O Alizadehkhaiyat, M Roebuck, G Kemp S Frostick, 15th EFFORT Congress, London, UK, 4-6 June, 2014,

- Correlation of the movement deviation profile of shoulder muscle EMG with measures of shoulder function, G Barton, D Hawkes, O Alizadehkhaiyat, A Howard, M Roebuck, A Fisher, S Frostick, M Robinson, M Hawks, European Society of Gait Analysis for Adults and Children, Stockholm, September, 10-15, 2012.

National – Podium

- Clinical Tests and their relations to fMRI, A Howard, J Gibson, O Alizadehkhaiyat, S Frostick, British Elbow and Shoulder Society, Sheffield, 24 June 2015.

National – Poster

- Muscular coordination during arm elevation: defining the balance between the deltoid and rotator cuff, D Hawkes, A Howard, O Alizadehkhaiyat, M Roebuck, S Frostick, BOA, Manchester, 11-14 September 2012.

- Muscle Patterning in Patients with Complex Shoulder Instability, A Howard, D Hawkes, O Alizadehkhaiyat, J. Gibson, G Kemp, S Frostick, Joint Meeting of the Bone Research Society and the British Orthopaedic Research Society in Oxford, 4-5 September, 2013.

- Muscular coordination during arm elevation: defining the balance between the deltoid and rotator cuff, D Hawkes, A Howard, O Alizadehkhaiyat, M Roebuck, S Frostick, BOA, Manchester, 11-14 September 2012.

Local

- Poster - What functional magnetic resonance imaging (fMRI) tells us about Complex Shoulder Instability, Academy of Medical Sciences, Leeds, 23 May 2014

Papers

- Muscle strength and its relationship with skeletal muscle mass indices as determined by segmental bio-impedance analysis, O Alizadejkjalyat, D Hawkes, G Kemp, A Howard, S Frostick, Eur J App Physiol, 2014, 114(1) 177-85.

- Correlation of the movement deviation profile of shoulder muscle EMG with measurer of shoulder function, G Barton, D Hawkes, O Alizadehkhaiyat, A Howard, M Roebuck, A Fisher, S Frostick, Gait & Posture 11/2013 38

Papers in preparation

- Is Shoulder Instability all in the mind – A complete white and grey matter analysis – target journal: Nature Neuroscience

- Movement deviation profile of shoulder muscle EMG of patients with Polar II/III instability – target journal: Eur J App Physiol

- Using movement deviation profile to define shoulder pathology – target journal: Gait & Posture

Abbreviations

AD	Anterior Deltoid
BB	Biceps Brachii
BOLD	Blood Oxygen Level Dependent
CIMT	Constraint-induced movement therapy
CSF	Cerebral Spinal Fluid
EMG	Electromygraphy
FDR	False Detection Rate
fMRI	Funtional Magnetic Resonance Imaging
FWER	Family Wise Error Rate
ISP	Infraspinatus
LD	Latissimus Dorsi
LD Inf	Latissimus Dorsi Inferior
LD Sup	Latissimus Dorsi Superior
MC	Motion Capture
MD	Middle Deltoid
MDI	Multiple Directional Instability
MRI	Magnetic Resonance Imaging
MVC	Maximum Voluntary Contraction
OIS	Oxford Instability Score
PD	Posterior Deltoid
PM	Pectoralis Major
PM Inf	Pectoralis Major Inferior

PM Sup	Pectoralis Major Superior
rTMS	Repetitive transcranial magnetic stimulation
SA	Serratus Anterior
SEM	Standard Error of Measurement
SLAP	Superior Labral Anterior and Posterior Lesion
SPM	Statistical Parametric Mapping
SSP	Supraspinatus
SUB	Subscapularis
TM	Teres Major
TPM	Tissue Probability Map
TR	Time Resolution
UT	Upper Trapezium
WOSI	Western Ontario Shoulder Instability Index

1 Introduction

1.1 Research Questions

1. Do patients with complex shoulder instability exhibit different cortical activation whilst using their shoulder, and if so, what are those differences?

2. Do patients with complex shoulder instability exhibit different muscle patterning whilst using their shoulder and if so, what are those differences?

1.2 Hypothesis

Patients with complex shoulder instability exhibit different cortical activations and muscles patterning whilst using their shoulder and these correlate with clinical measures of shoulder instability.

1.3 Objectives

1. To develop a study protocol for the assessment of cortical and muscle activation patterns in patients with unstable shoulders, using EMG and fMRI.

2. To recruit patients with Polar type II/III shoulder instability and healthy volunteers into the study.

3. To measure the cortical activation of both patients and controls whilst undertaking upper limb movement using fMRI.

4. To measure muscle activation of the patients and controls whilst undertaking upper limb movement using EMG.

5. To analyse the data appropriately and draw conclusions from the healthy volunteers compared to the patients with Polar type II/III.

1.4 Defining the Clinical Condition of Shoulder Instability

The shoulder joint has a large humeral head compared to glenoid, and a large degree of stability is achieved from the soft tissues. The concept of shoulder instability is explored in detail in Chapter 2.1. Simply put, shoulder instability is an inability to maintain the humeral head in the glenoid fossa, associated with discomfort, slipping or a sense that the shoulder is unstable and could dislocate [1].

There are many different systems to classify shoulder instability that are explored later in this chapter; however the Stanmore classification [2] is the more accurate at defining the patient group in this study (Figure 1.1). The patient group falls along the Polar type II to Polar type III continum. Instability in this group of patients is derived from a combination of muscle patterning issues and atraumatic structural defects in different proportions.

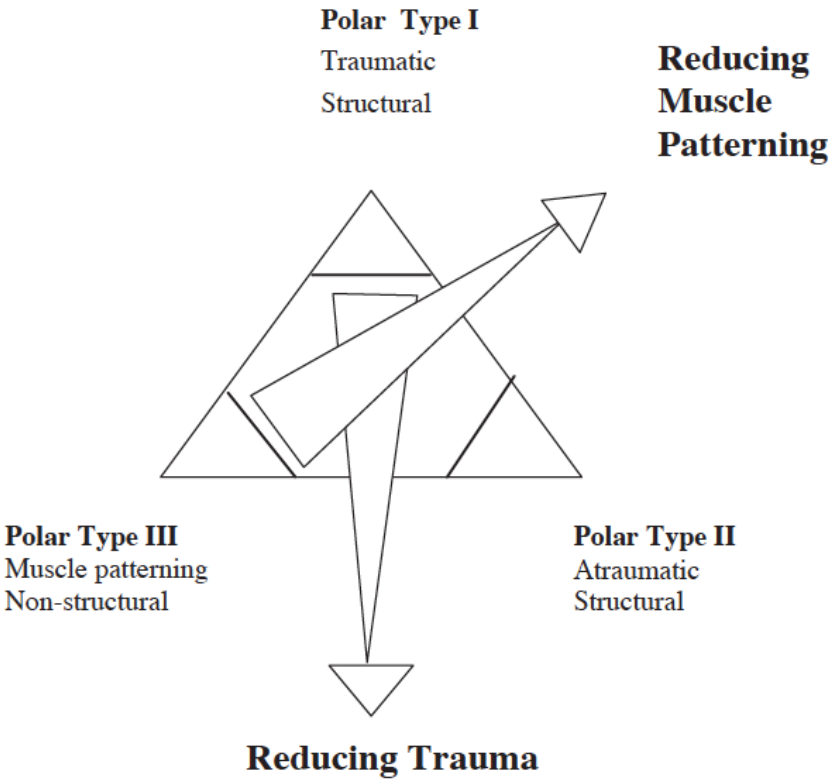


Figure 1.1 – Diagram showing the Stanmore classification, which classifies shoulder instability into three aetiology Polar groups, demonstrating the continuum nature of this shoulder condition [2].

1.4.1 Principles of Modalities

The following sections will outline the main principles of the data collection and processing. The modalities used in my work are dealt with in detail in the literature reviews. This section sets out an introduction to the techniques and how data processing was approached.

1.4.2 Functional MRI

1.4.2.1 Basic Principles

The basis of fMRI can be traced back to the physiologist Charles Sherrington in 1890, who demonstrated that brain activity caused a local increase in blood flow [3]. BOLD fMRI relies on the property that an increase in neuronal activation causes locally increased ratio of oxy- to deoxyhaemoglobin, which can be used to develop image contrast. Thus the deoxyhaemoglobin is an indirect marker of neuronal activity in the grey matter of the brain, as was initially demonstrated in a cat brain [4]. The relationship is complex, with neuronal activity being both excitatory and inhibitory [5].

The signal contrast is generated by the change in magnetic susceptibility: haemoglobin bound to oxygen is diamagnetic, whilst

deoxygenated haemoglobin is paramagnetic. This produces the detectable contrast [6], as demonstrated by Thulborn et al. [7].

The temporal and spatial resolution of the BOLD response are important concepts. The temporal resolution determines detection of the time-dependent BOLD response to the stimulus (Figure 1.2): following stimulus there is a peak until the stimulus is removed, whereupon there is an undershoot until it recovers back to the baseline. Spatial resolution refers to the fact that the haemodynamic response is not spatially very specific to cortical activity, although this is less of an issue in the motor and sensory cortex [3].

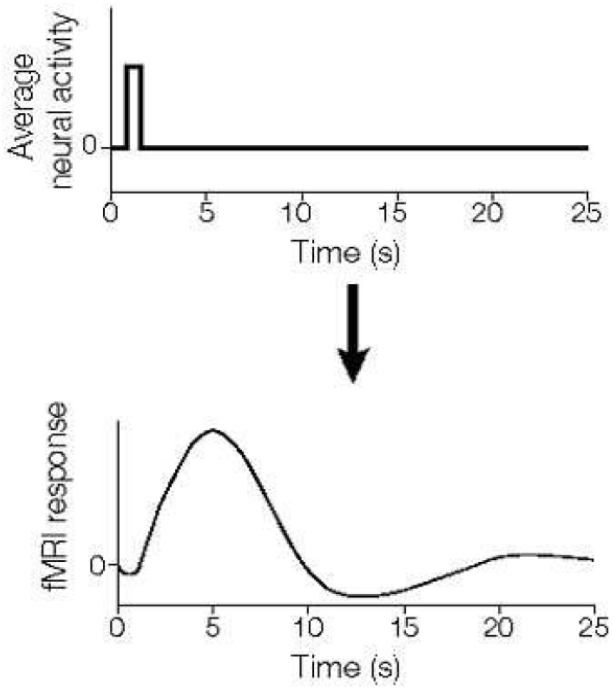


Figure 1.2. Diagram to demonstrate the haemodynamic response for fMRI data [8].

1.4.2.2 Data Processing

There are a number of steps to prepare the raw data in order that statistical analysis can be undertaken. The steps outlined below (Figure 1.3) were implemented in SPM12 (UCL, London) [9].

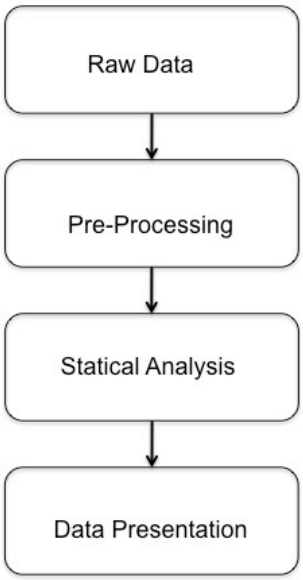


Figure 1.3. Diagram to show the steps in data processing for fMRI.

The aim of pre-processing is to ensure that in the statistical analysis we are confident that we are comparing voxels in the same location. The pre-processing steps can be divided as follows:

i. *Slice Timing correction* – The echo-planar scanning sequence used has a TR of 3 seconds, which means that it took 3

seconds for all the slices of the brain to be acquired. We need to correct the data for this delay to regain the time course that presents the data for the whole brain at the same point. This is achieved by interpolating between the actual point of data acquisition and a single point in time achieved through Fourier transformation [10].

ii. *Realign & Unwarp* – Realignment is the correction for head movement, in an attempt to align the slice to the one before, thus to be confident that activation occurs in known anatomical areas. Movement in our paradigm was potentially an issue, however a deliberate decision was not to use task-correlated motion as there was a risk of type 1 errors [11]. The brain is not homogenous, the structures themselves induce distortion which cause warping of the real image [12]. The six movements calculated in the realignment calculations used in conjunction with a static deformation field map are used to estimate the deformation.

iii. *Segmentation* – This step separates out the different tissue types, CSF, grey matter, white matter and bony skull; bias corrects and spatially normalises [13]. The six different tissue probability maps within SPM12 were averaged in a flipped and un-flipped orientation for all subject activations to be converted to the right-sided movement.

iv. Co-registration – This again attempts to account for motion but analyses the movement between the functional scans and the T2 structural scans. This uses a concept of "mutual information" to establish the probable relationship between voxel intensities and attempts to maximise that intensity [14].

v. *Normalise* – An algorithm is used to transform each subject's brain into a standard space that enables comparisons to be made across subjects. SPM undertakes this process in two stages, linear and then non-linear Bayesian transformation [15].

vi. *Smooth* – This acts as a low pass filter, removing the high frequencies of the signals enhancing improving the low frequencies and sensitivities. The fMRI signal is convolved with a Gaussian function of a defined specific width, expressed as Full Width at Half Maximum. As the analysis contained greater than 16 participants, smoothing [8 8 8] was used [16]. In essence a voxel is averaged compared to those that are adjacent and there is a blurring of the edges improving the spatial correlation [17].

vii. *Further Motion Correction* – A major source of artefact is head movement, this is particularly acute in a paradigm that

requires movement of the shoulder. The original six degrees of freedom calculations were used as a covariance of no interest when constructing the contrast at the first level.

1.4.3 Electromyography

1.4.3.1 Basic Principles

EMG is a measure of the action potentials generated by muscles, which causes muscle contraction [18]. Electrodes placed on the skin or within the muscle detect the action potential signal. As the signal collected is only in millivolts, these signals amplified using pre-amplifiers. The pre-amplifiers are a short distance from the electrodes which reduces the noise within the EMG signal [19, 20], Chapter 3.4.2.1.b.

At a cellular level, the muscle movement starts when there is an influx of ions causing the depolarisation along the muscle fibre, known as an action potential [21]. The depolarisation is shown in Figure 1.4, which demonstrates how the amplitude and the direction change as the action potential travels under the two surface electrodes. At point T2 and T4, the potential difference between the two electrodes is the greatest; the first is positive and the later negative.

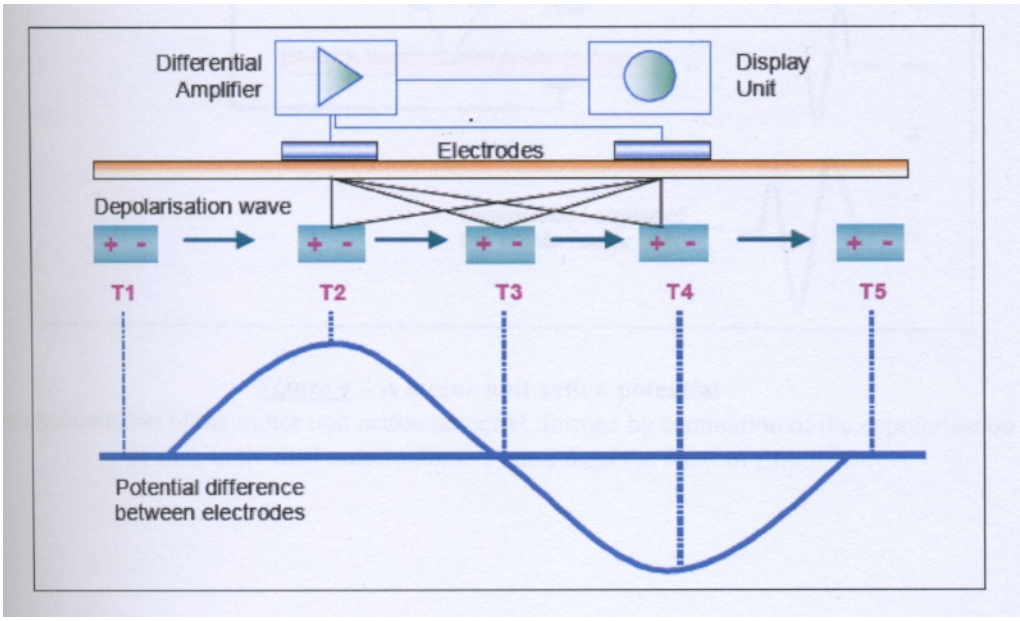


Figure 1.4. The diagram shows the depolarization along a single muscle fibre [21, 22].

The picture is more complicated, as the above represents only a single muscle fibre; there will be many motor neurons innervating vast numbers of muscle fibres within a single muscle. Thus the surface electrodes will detect a sum of all the action potentials.

The amplitude of the signal is proportionate to the degree of activation of the muscle, but there are complicating factors that make the relationship variable and non-linear [23]. Thus muscles activated at the same time and in similar locations can produce different activations which are non-linear [24]. These inter-muscular differences can be caused by differences in firing rate properties, motor unit recruitment, agonist-antagonist muscle interactions or cross talk from two adjacent muscles [23].

The picture is further complicated by the fact that in movement, particularly in the shoulder, muscles change location relative to each other and to the electrodes. The movement is associated with changes in the muscle length, relative fibre movement and changes in the relative amplitude, all of which influence the action potential sensed by the electrode.

1.4.3.2 Data Processing

As with the fMRI data collection a number of preprocessing steps had to be undertaken, these can be divided into the following processes:

i. *Rectification* – The raw data is both positive and negative, and this process inverts all the negative values to positive [21].

ii. *Smoothing* – The raw data fails to capture all the activations and does not represent all the motor unit recruitment, producing a random signal. Two mathematical algorithms (moving average and root mean square) can be used to calculate the average values and thus the trends within the raw signal. The root mean square was used.

iii. *Amplitude Normalisation* – The signals are variable, influenced by location conditions, such as temperature, subcutaneous fat, perspiration; operator conditions, such as electrode placement and activities related such as muscle fatigue [25-27]. In order to compare the values the signals have to be normalised, for which a number of methods can be employed. One method would be to record MVC and normalise to this value. However, this was inappropriate in my study as the pain has been shown to produce an invalid MVC value [28]. The validity of a isometric contraction for a dynamic contraction has been questioned [29] and individual training/motivation has been shown to produce variability in results of between 10-30% [30, 31]. Most of the patient group suffered pain on shoulder movement, and there was a desire to standardise normalisation consistently across all patients. Following similar studies involving cyclical dynamic movements, the mean value was been used [32-34].

iv. *Time Normalisation* – Throughout the various protocols, a great deal of variation in movement was possible. This variation is particularly high in the shoulder, which has six degrees of movement. Thus for each of the muscles 10 cycles of movement were averaged to give a more representative movement characteristic. Each phase of a

movement was converted to a scale of 100%, a method employed in similar studies [35].

In the next three sub-chapters, there will be an exploration of the literature around shoulder instability and fMRI/EMG work that relates to the shoulder.

2 Literature Review

In the following reviews of literature in the next three sub-chapters, it is my intention to give examples of research that relate directly to my thesis, rather than provide an encyclopedic narrative.

2.1 Shoulder Instability

This section will focus on the shoulder instability that relates to muscle patterning, although in order to understand the context of this condition it is necessary to review other overlapping shoulder pathologies.

2.1.1 Classification of Shoulder Instability

Hippocrates first described shoulder instability in 460 BC. He described the treatment by the insertion of a “Red Hot” iron into the axilla, which caused scarring in the lower part of the joint as a treatment for recurrent instability following an acute dislocation [36].

A seemingly basic question “what is shoulder instability?” is deceptively complex if considered fully. There is a degree of translation of the glenohumeral joint in normal shoulder movement. However, shoulder instability can be defined as the lack of ability to

maintain the humeral head in glenoid fossa, without discomfort and "a feeling of looseness, slipping, or the shoulder 'going out'" [1].

Ambiguity in the classification of shoulder instability has long been recognised as leading to the failure in treatment of specific pathologies [2, 37, 38]. For the clinicians, it is imperative to understand not only the condition but also the classifications. Success in both will lead to appropriate treatment.

Bankart [2] introduced the concept of unidirectional anterior instability, caused by a Bankart lesion, described in 2.1.1.2. In unidirectional instability, the defect is in the inferior labrum causing capsule laxity enabling anteroinferior dislocation. Neer at al.[39] subsequently introduced the concept of multidirectional instability, describing patients who in addition to anterior instability have a component of inferior instability and posterior instability.

The Rockwood [40] classification used trauma as the guiding principle in defining instability (Table 2.1). The Thomas and Matsen classification suggested that trauma was the greatest etiological determinant, dividing the patients into two groups: those with **t**raumatic **u**nidirectional **B**ankart lesion treated with **s**urgery (TUBS), and **a**traumatic **m**ultidirectional **b**ilateral treated with **r**ehabilitation and if surgery is required an **i**nferior capsular shift (AMBRI) [41]. However, the Thomas and Matsen classification is silent to the

voluntary instability group who do not feature in their classification. Schneeberger and Gerber[42] which further developed both the classifications of Rockwood & Thomas and Matsen. Again their system is based on trauma being the only causative agent, which ignores patients with voluntary instability and it does not address the role of generalised laxity in shoulder instability [2].

Table 2.1 – Table to show the division of the Rockwood Classification of shoulder instability [40].

Type I	Traumatic subluxation without previous dislocations
Type II	Traumatic subluxation after previous dislocations
Type III	Atraumatic voluntary subluxation (A) With psychiatric problems (B) Without psychiatric problems
Type IV	Atraumatic involuntary subluxation

It had been observed that shoulder instability is a dynamic process and patients who present with muscle patterning issues may develop structure issues [43]. This continuum multicausal approach was the basis of the Stanmore classification, defining three polar groups (Figure 1.1): Polar type I, traumatic/structural; Polar type II, atraumatic structural; Polar type III muscle patterning non-structural, which can be further sub-divided (Figure 2.1). The focus of this thesis is on a patient group who fall on the line between Polar type III, muscle patterning non-structural and Polar type II, atraumatic structural.

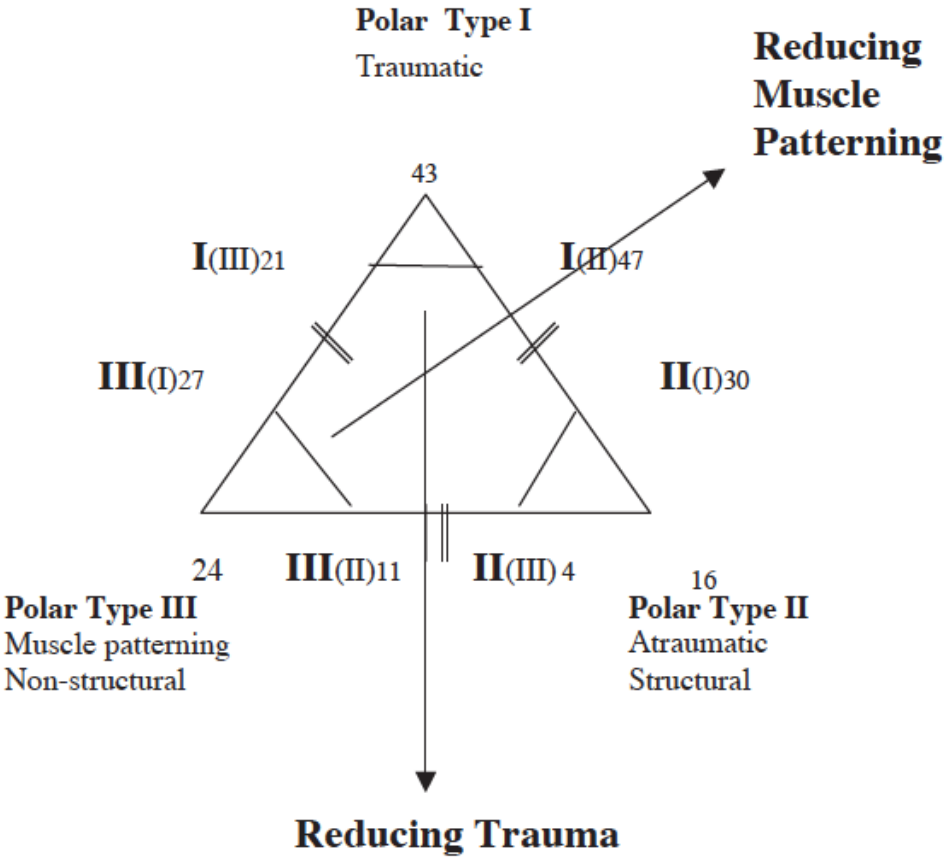


Figure 2.1 – Diagram showing the sub-divisions of the Stanmore classification, the numbers in superscript show a retrospective analysis of referrals received by the Stanmore Institute over an undefined period, n=223 [2].

The characteristics of the three groups enable patients to be objectively described, but the Stanmore classification enables patients to be described in a manner that portrays the reality that these shoulder pathologies exist on a continuum (Table 2.2 A). The most common group is Polar group type I, traumatic instability. In dislocations with up to 95% of patients under 20 years and 14% over

40 years of age will present with instability [44]. It can be seen (Table 2.2 A) that Polar type III characteristics relate to muscle patterning, laxity and capsular dysfunction. In contrast, Polar type II represents a hybrid between Polar types I and III, with the underlying characteristic atraumatic structure defects. The further subdivisions (Table 2.2 B, Figure 2.1) reflect the etiological disparity of the original patient group (number 223), however these are clinically unhelpful. What they highlight is the fundamental strength of this classification in its comprehensive nature and the way it exemplifies that these conditions exist on a spectrum.

Table 2.2 –Table demonstrating the characteristics of the polar groups of the Stanmore triangle, A and the characteristics of the sub-classification of the polar groups, B [2].

A.

Pathology	Group I	Group II	Group III
Trauma	Yes	No	No
Articular surface damage	Yes	Yes	No
Capsular problem	Bankart lesion	Dysfunctional	Dysfunctional
Laxity	Unilateral	Uni/bilateral	Often bilateral
Muscle patterning	Normal	Normal	Abnormal

B.

Pathology	Group I (II)	Group II (I)	Group I(III)	Group III(I)	Group II(III)	Group III(II)
Trauma	+++	++	++	+/−	+/−	+/−
Articular surface damage (Humeral head and/or gleniod rim)	Yes	Yes	Yes	No	Yes	Yes
Muscle patterning	No	No	Yes	Yes	Yes*	Yes

**BUT apparent on functional EMGs.*

Kuhn et al. undertook a systematic review of the factors that the various classification system employed (Figure 2.2) and developed

their own classification system (Table 2.3), named “FEDS”. As will be apparent from the questions, the classification system relies on the patient’s perception of their condition, which is a weakness given the frequent difference in individuals’ own perception.

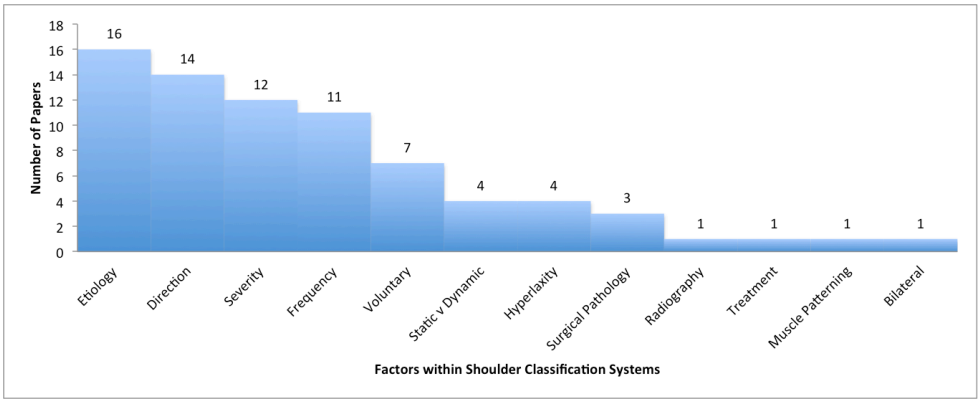


Figure 2.2 – Graph to show the factors that made up the following classification system, following Kuhn et al. [1] Systematic review: Allen [45], Cole and Warner [46], Corfield and Irving [47], Galinat and Warren [48], Gerber and Nyffeler [49], Joseph et al. [50], Lewis et al. [2], Maruyama et a. l[51], Nebelung, Ozkan et al. [52], Pollock and Flatow [53], Protzman [54], Rockwood [55], Schneeberger and Gerber [42], Sillliman and Hawkins [56], Thomas and Matsen [41], Wirth and Rockwood [57].

Table 2.3 – Table setting out the questions that make up the FEDS classification for shoulder instability. FEDS encompasses, Frequency, Etiology, Direction and Severity [1].

FREQUENCY–The patient is asked, *"How many episodes have you had in the last year?"*

Solitary – *'1 Episode'*
Occasional- *'2 -5 Episodes'*
Frequent – *'>5 Episodes'*

ETIOLOGY – The patient is asked, *'Did you have an injury to cause this?'*

Traumatic – *' Yes'*
Atraumatic – *' No'*

DIRECTION – The patient is asked, *' What direction does the shoulder go out most of the time?'*

Anterior- *'Out the Front'*
Inferior- *' Out the Bottom'*
Posterior- *'Out the Back'*

The direction is confirmed at the time of the physical examination using provocative tests. During translation testing, the physician asks, which one of the following directions most closely reproduces your symptoms, and then translates anterior, inferior, and posterior. To confirm, the physician may ask which one of these tests most closely reproduces your symptoms: and the anterior apprehension test, the sulcus test, and the posterior jerk test is performed. With the history and physical examination using provocative tests, the patient should be able to distinguish and identify the ***primary direction*** of his or her instability.

SEVERITY–The patient is asked,*'Have you ever needed help getting the shoulder back in joint?'*

Subluxation– *'No'*
Dislocation – *'Yes'*

It is clear that there is a psychological aspect to the condition. Anecdotally, I have seen cases where there is strong evidence that it is a factor at play in a patient's shoulder instability. For example, a child approaching important exams developed shoulder instability and recurrent dislocations; because of the instability, the child's parents deferred the exam for a year, at which point the instability resolved almost instantly and spontaneously (personal observation). Rowe et al. [58] in 1973 published a series of patients who voluntarily dislocated their shoulder, finding that after psychological

testing those who had not experienced psychological issues improved with treatment, whereas those who did responded poorly. Shoulder research has used terms such as habitual subluxation of the shoulder [59], voluntary instability [60] and involuntary positional instability [61]. Takwale et al. classified their patient group into those with and without psychological disturbance, however they failed to define the term [61].

2.1.1.1 Epidemiology

There is high prevalence of shoulder disorder amongst the population, with up to 50% of the population experiencing at least one episode of shoulder pain annually and high recurrence rates of up to 54% [62]. Laxity of a general nature occurs in 4.2 - 4.6%, however it is important to appreciate that laxity is not synonymous with instability [63]. It is thought that glenohumeral instability is present in 2% of the population, though this is influenced by the activities engage in by the patient; for example, in athletes it is around 10% [2].

Instability often occurs in response to dislocation; 96% of dislocations occur due to trauma and 4% are atraumatic resulting from repetitive use or minor injury [64].

The age of onset for the instability is higher in the younger population, 44% below the age of 40 years, compared to those above which is 11% [65].

2.1.1.2 Pathophysiology

The pathophysiology of shoulder instability is complex with causal factors at different levels, gross anatomy, subdermal, cellular and genetic. I will focus on the structural aspects of the bone/soft tissue of the shoulder and their control.

The Stanmore classification features two types of mechanisms causing structural defects, traumatic, Polar type I and atraumatic, Polar type II. I will firstly consider the nature of these defects before then addressing the pathophysiology of polar type III. As implied by the triangle, a patient may possess varying amounts of all three components of the polar groups.

The following is a summary of the structural defects that can be persistent in either polar type I or polar type II:

i. Bankart Lesion [59, 66], which is a detachment from the glenoid rim of the capsuloligamentous complex (Figure 2.3). This causes the disruption of the structural integrity of the capsule, causing potentially further dislocation and/or pain;

ii. Hill-Sachs Lesion [67], (Figure 2.4), where an anterior dislocation causes an impression fracture of the posterolateral humeral head as it comes in contact with the glenoid rim during the dislocation and subsequent relocation.

iii. Superior labral anterior and posterior lesion [68, 69], known as a “SLAP lesion”. This is a pathological abnormality of the glenoid labrum, which extends from the posterosuperior to the anterosuperior aspect of the glenoid (Figure 2.5). Further, it can be subdivided into simple degenerative changes at the free edge of the labrum (Type I), to lesions involving the displacement of the labrum into the actual joint (Type IV) [70].

iv. Attenuation of the capsular ligaments [71], which is a reduction of the strength of the capsular ligaments leading to greater translation of glenoid head, leading to dislocation.

v. Disruption of the subscapularis tendon (Figure 2.6) [72-75].

vi. Humeral avulsion of the inferior glenohumeral ligaments [76].

Figure 2.3 – Diagram showing a Bankart Lesion [77].

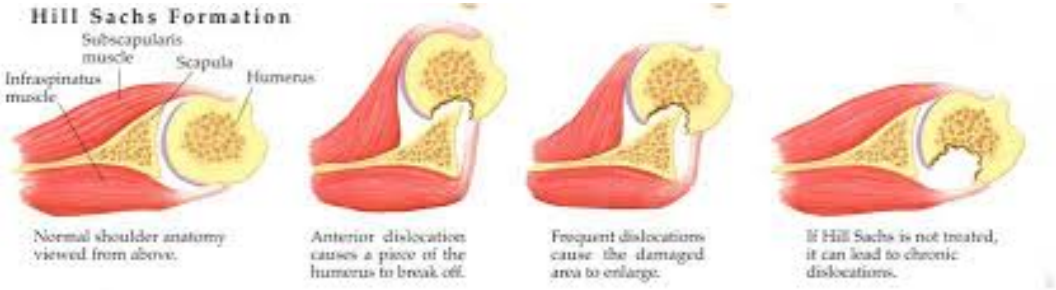


Figure 2.4 – Diagram to show how an anterior dislocation causes an impression fracture of the posterolateral humeral head.

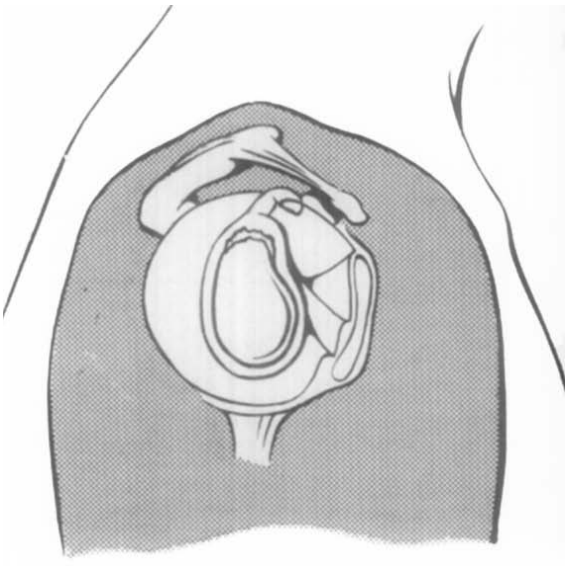


Figure 2.5 – Sketch showing a Superior labral anterior and posterior or a SLAP lesion [70].

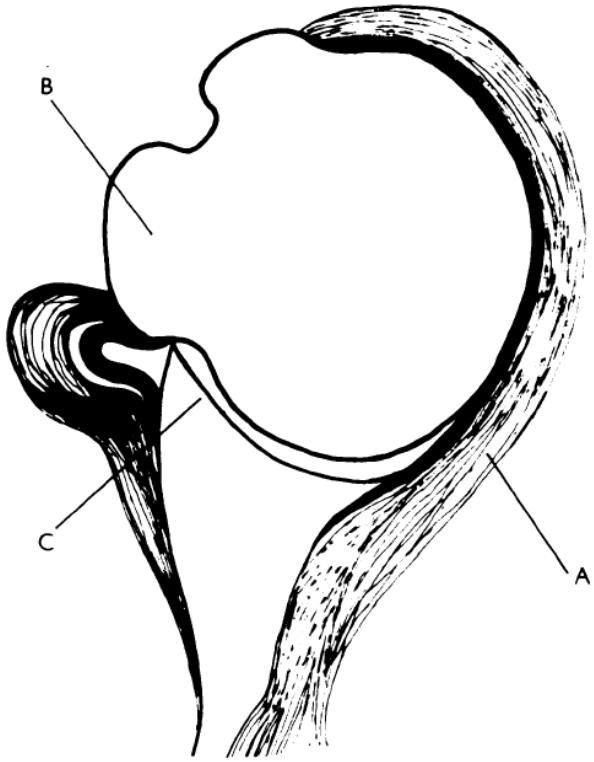


Figure 2.6 – Sketch showing a horizontal section of left shoulder demonstration the location of the subscapularis muscle (A), greater tuberosity (B) and glenoid fossa (C). The sketch demonstrates when the muscle is in tension it comes into contact with the posterior margin of the glenoid fossa [75].

Accurate identification of the patient's pathoanatomy is critical in providing effective treatment [37]. Focus will remain on Polar II and Polar III type patients, given the patient group being investigated in this thesis. As physical defects have already been explored above, the issues of muscle patterning will now be addressed.

In polar type II, the glenoid has an abnormal translation, the anterior translation, which is a result of one or more of the following factors: congenital labral pathology, excessive anterior capsular laxity, scapular dyskinesis, muscular imbalance [64]. In some patients, for example throwing athletes, their over-use generates a muscle imbalance [78]. However, for the majority of our patient groups the pathogenesis is not so clear and thus it is important to understand potential causes of instability. Polar type III patients often develop shoulder instability following a trivial injury [79], which is the case with the majority of the patients in this study. Although the level of trivial injury may pull them towards the Polar type I, part of the spectrum or congenital structural abnormalities push them towards the polar type II group.

It has been known that dysfunctional activation patterns cause shoulder dislocation, the polar type III patients. However, given the large range of movement and the changing stabilising forces over

the course of that movement, it is difficult to tease out the muscle forces that generate the instability and/or dislocation [80].

Kido et al. [81] established that the anterior deltoid has an anterior stabilising role in abduction and external rotation, and this role becomes increasingly important as the shoulder became unstable. The posterior translational force is resisted by subscapularis and this muscle along with supraspinatus were particularly important in the mid-range movements [82]. Excessive humeral head excursion has been observed in instability patients [83]; the muscles govern to a large extent the position of the humeral head in the glenoid fossa.

In patients with instability the activity of three parts of deltoid is decreased (10-38%) whilst the activity of the rotator cuff muscles is increased (15-20%). Illyes et al. further found that the length of activity in the instability patients of pectoralis major/deltoid was shorter in supraspinatus, infraspinatus and biceps brachii, possibly a compensatory mechanism to centralise the glenohumeral head [84]. These findings were consistent with Kronberg et al. work, who 15 years previously found a similar pattern in both forward flexion and abduction [85]. However, both these studies contradicted the findings of McMahon et al. in their work comparing anterior glenohumeral instability versus controls. In respect of forward flexion and abduction, less activity was seen in serratus anterior in the instability patients but no muscles (subscapularis, supraspinatus,

infraspinatus, rhomboids, serratus anterior and trapezius) demonstrated a significant difference between controls and patients. Dysfunctional scapulohumeral rhythm has been shown in instability patients, with differences in the scapular protraction and/or spinal tilt due to inadequate muscular activity. This then changes the alignment of glenohumeral joint predisposing the individual to shoulder instability [86]. As discussed in Chapter 2.1.1.6, scapular position features in physiotherapy treatment for patients with instability who are being managed conservatively.

Jaggi et al. in their retrospective study, advanced the theory that latissimus dorsi and pectoralis major are the two muscles predominantly involved in instability, but acknowledged that further studies were required [87]. Latissimus dorsi was found to be inappropriately active in both anterior and posterior instability, with pectoralis major more active in anterior instability. Further, they found that infraspinatus was inactive in posterior instability. This work confirmed the cadaveric work of Konrad et al. [88], which considered the joint reaction forces (Figure 2.7) and the role of the shoulder muscles that are critical to the stability of the shoulder. In cadaveric work on latissimus dorsi and pectoralis major, Pouliart and Gagey [89] drew the following conclusions:

i. Both decreased the maximum angle of shoulder abduction and external rotation.

ii. They increased the anteriorly directed joint reaction force, which leads to anterior translation.

iii. Instability could be generated by both these muscles dependent on the joint angle

Pectoralis major dysfunction has been identified as particularly important at the extremes of movement in both flexion and abduction [90]. In work with multidirectional instability and multidirectional laxity, Morris et al. demonstrated that muscle activation was dysfunctional in anterior and posterior deltoid. In the former group, the anterior deltoid activation was different at 90 and 0 degrees of abduction whereas posterior deltoid activation was different during rotation at 90 degrees. In the latter group, there was increased activation in posterior deltoid during abduction [91].

In cadaveric work Blasier et al. found that subscapularis had the greatest subluxation force [92]. The importance of the coordination of muscles to maintain shoulder stability were reinforced by Kronberg et al., who further concluded that infraspinatus, subscapularis and latissimus dorsi acted as important stabilisers during flexion of the upper limb [93].

In patients who were able to voluntarily dislocate their shoulder whilst undergoing EMG analysis, it was found that posterior dislocation was achieved with activation of infraspinatus/posterior deltoid, whereas anterior dislocation was achieved with activation of anterior deltoid and bicep muscles [94]. The majority of polar type III patients presented with posterior instability [43, 61].

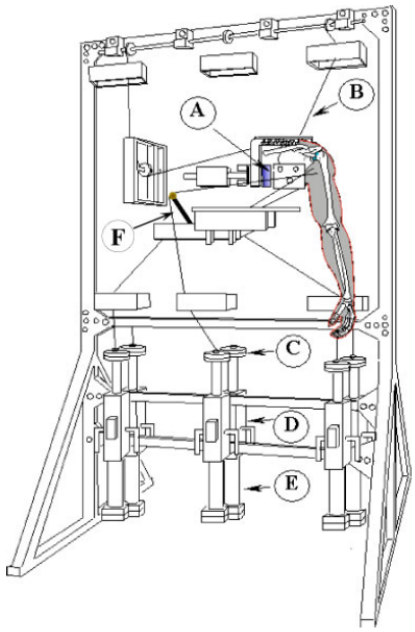


Figure 2.7 – A sketch showing the dynamic shoulder testing apparatus: (A) universal force-moment sensor attached to the scapular mount; (B) cable-pulley system; (C) load cell; (D) hydraulic cylinder; (E) linear variable differential transducer; (F) cable-pulley system for pectoralis major [88].

Deficiency in proprioception has been identified in a number of movement disorders, and proprioception is essential for the co-ordination of multiple joint sequences, such as movement of the upper limb. Barden et al. found that compared to controls there was a deficiency, as measured in a repeated hand measure.

Interestingly they found the degree of error improved during testing and there was no difference between the symptomatic and asymptomatic instability patients [109].

A more complex and controversial area of pathophysiology is instability that is seen as resulting from a psychological cause. These range from voluntary “trick” movements, where abnormal muscles are recruited to dislocate the shoulder [58, 60, 95] to involuntary recruitment of abnormal couples generated in a variety of movements or positions.

Rowe, Peirce and Clarke [58], describing voluntary subluxation, found some degree of psychiatric abnormality in 30% of their patient group, although this was refuted by Huber and Gerber [60]. However, as mentioned above the role of a psychological component has never been properly assessed, and therefore its causative role in unknown.

2.1.1.3 Diagnostic Evaluation

Recording historical information is critical in the diagnostic process [96]. It has been found that with a good history and clinical examination, the correct diagnosis is possible in 90% of instability patients [97].

Whilst there may be clinical signs of laxity, namely the Sulcus sign (Figure 2.8), the critical tests for instability are the anterior and posterior apprehension tests. Kumar et al. has established that these are 96% sensitive and may be superior to MRI investigation in certain circumstances. However, the assessment of polar type II and type III demands greater observation, dynamic observation for scapular winging is important. Scapular dyskinesis after repetitive movements may be indicative of fatigue failure of the external rotator muscles [98], however others suggest that this is due to decreased trunk stability and over activation of pectoralis major [64]. In addition to examination of the shoulder, there needs to be an assessment of the kinetic chain and overall assessment of posture.

Figure 2.8 – Photograph showing the Sulcus sign [64].

The plain radiograph has a limited role in diagnosis of shoulder instability, however it may be helpful in identifying Hill Sachs defect [67] which is pathognomonic of structural anterior instability [99]. Unless evaluating instability against a background of trauma, MRI has a sensitive up to 93% [100].

Although examination under anaesthetic has a high sensitivity [101], the mainstay of treatment it is an arthroscopy. This modality is particularly helpful if there is no firm evidence of dislocation and no definitive diagnosis can be reached through less invasive diagnostics. Mok et al. established that diagnostic arthroscopy it was 80% sensitive and changed diagnosis in 20% of cases in their case series of 166 patients. These patients had symptoms of subluxation without any history of traumatic injury or firm diagnosis [102]. Lewis et al. states with reference to the Stanmore triangle that arthroscopy is fundamental to differentiate between Polar type II/atraumatic structural and Polar type III/muscle patterning non-structural shoulder instability.

Although EMG has been used in the clinical setting since the 1970's, its use is limited to specialised shoulder units and even then availability is limited [87]. However, what is critical is that any muscle patterning issues are identified and these may not be readily apparent. Failure to endeavor to correct these issues has been

linked to failure of subsequent surgery, particular in polar type III patients [103].

2.1.1.4 Risk Factors

Risk factors can be divided in to intrinsic and extrinsic factors

Extrinsic Factors:

a. *Dislocation*. The mechanism variety is limitless, however, shoulder instability secondary to the following mechanisms has been established; collision sports, assaults, seizures, motor vehicle accidents [104, 105].

b. *Occupation*. Any occupation which involve tasks being undertaken about chest height has been shown to increase the risk of developing shoulder instability [106].

c. *Primary damage or Secondary to trauma*. Bankart lesion [59, 66], Hill-Sachs lesion [67], superior labral anterior and posterior lesion [68, 69], attenuation of the capsular ligaments[71], disruption of the subscapularis

tendon [72-75], humeral avulsion of the inferior glenohumeral ligaments [76].

Intrinsic factors:

a. *Hyper-mobility*. Patients with collagen disorders such as Ehlers-Danlos syndrome or hyper-mobility syndrome have a three-fold increase risk of shoulder instability [65, 87, 107-109].

b. *Age*. Grouping the previous studies [104-106, 110-116], those patients who had undergone a primary dislocation, instability precipitating further dislocations were higher in those below 40 (44%), compared to those above (11%) [65]. These studies are limited to traumatic dislocations, rather than instability as a result of another sequel.

c. *Sex.* Although the meta-analysis of Olds et al. suggests there is a three-fold increase in men suffering from recurrent instability, their inclusion of lower quality studies means there is a need to treat this assertion with caution. There is a mixed picture when sex is examined at different age ranges.

2.1.1.5 Prognostic Factors

If the instability is precipitated by a traumatic dislocation, then age plays an important determinant on prognosis, those below 40 years of age are 13.5 time more likely to suffer recurrent instability [65].

Apart from trauma, the activity that has precipitated the instability is predictive of recovery. In patients who developed instability following participation in overhead throwing sports, the rate of spontaneous recovery was 8.7 times higher after ceasing the activity compared to those that continued; however, if the precipitating cause was non-overhead sports, then spontaneous recovery after ceasing the activity fell to 1.4 times higher compared to those who continued to play [43].

In terms of polar group III and polar group II instability, there is a notable absence of research to support prognostic estimates. However, in instability patients being treated conservatively, 60-70% demonstrated reduced instability [117, 118]. Whilst this sounds reassuring at the point of initial diagnosis, at the end of treatment in the polar type II or III with no correctable structural deformity, it leaves 30-40% who will remain resistant to treatment. This

treatment-resistant group makes up a significant part of the patient group in this study.

2.1.1.6 Treatment

Treatment is dependent on the nature and cause of the instability, highlighting the need for an accurate diagnosis before treatment can commence. For patients with instability in more than one direction, treatment through a physiotherapy-led exercise program is the starting point [119].

Prior to the work of Putti, Bankkart, Platt and Bristow, treatment success of shoulder instability, such as it was defined, was low [2]. For Polar Type 1 patients, the treatment is dependent on the nature of trauma-induced defect. For example, if there is a Bankart lesion the detached labrum is restored by reattaching the Inferior glenohumeral ligament [120], and often the primary treatment is nonsurgical with supervised physiotherapy [121]. Recurrent traumatic dislocations may have stretched the capsule which may be surgically corrected through a number of techniques both open and closed [122, 123]. There a number of surgical options dependent on the nature of the pathogenesis; it is not intended to explore these further here, but Table 2.4 gives an overview of both the soft tissue and osseous procedures that are available [124]. However, there is

conflicting evidence with regards to outcomes for the wide variety of approaches shown in the table. For example, with regards to thermal capsulorrhaphy, Frostick et al. advocate this approach and after two years there have been no reported dislocations [121].

Table 2.4 – The various surgical options that are available in the treatment of glenohumeral defects [124].

Procedure	Consideration
Soft tissue	
Reverse Bankart repair (open or arthroscopic)	Often performed in combination with an arthroscopic capsular plication, posterior-inferior capsular shift, or reverse Putti-Platt
Arthroscopic capsular plication	Performed on patients with isolated unidirectional posterior instability without a true labral tear
Open posterior-inferior capsular shift	Surgical option for patient with posterior-inferior subluxation with no anterior component and a functionally intact rotator interval
Reverse Putti-Platt	Often reduces range of motion and is thus generally not recommended for athletes requiring full range of motion
Thermal capsulorrhaphy	Not recommended because of high recurrence rates
Osseous	
Posterior bone block or posterior wedge osteotomy	Generally indicated for patients presenting with a failed capsular plication, glenoid hypoplasia, increased glenoid retroversion, or an osteochondral fracture of the glenoid cavity vs posterior glenoid bone loss
McLaughlin's procedure or Neer's modification of McLaughlin's	Performed on patients with locked posterior shoulder dislocation resulting from reverse Hill-Sachs lesions encompassing 25% to 50% of the humeral head
Humeral head allograft	Alternative option to McLaughlin's or Neer's procedures based on the surgeon's preference/experience; our preference as the most anatomic way to reconstruct large engaging reverse Hill-Sachs lesions

The treatment of patients with Polar type II/atraumatic structure and Polar type III/non-structural muscle-patterning is more complex. The approach is to address the muscle patterning issues first, the position along the Polar II and III continuum is often difficult to identity. Kuroda et al. observed 573 patients with atraumatic instability within a minimum follow up time of 3 years. They

concluded that patients should be followed up for several years to avoid performing unnecessary surgery [43].

The conservative approach is aggressive physiotherapy aimed at strengthening the rotator cuff and the scapular stabilisers [125]. The success of biofeedback training and physiotherapeutic rehabilitation is high, with between 60-70% demonstrating reduced instability [117, 118].

Surgical intervention is only considered if there is a correctable structural abnormality either in the soft tissue or bone. For example, a glenoid with excessive retroversion and flatness may be treated with an osteotomy [126].

It has been proposed that 100% of voluntary instability can be successfully treated by early counseling to cease the movement [2]. This bold claim is substantiated by work done by Kruoda et al: “The important point with voluntary dislocation is that dislocation or subluxation does not occur once the patient is taught how to avoid it” [43].

From the prognostic section in this sub-chapter [2.1.1.5], in Polar groups II and III between 30-40% are resistant to treatment. As mentioned above this patient group makes up some of the patients examined during the course of my work.

Part of the basis for conservative treatment is providing visual feedback about motor performance [127], however recent techniques in treating shoulder instability have borrowed treatment methodology used in chronic pain syndrome and phantom limb rehabilitation [128-131]. Ramachandran [132] first used mirror therapy to provide feedback to patients with phantom limb issues of both pain and a sense that the absence limb was in an awkward position. The mirror was positioned in the sagittal plane to give the illusion that the absent limb had returned and patients experienced the sensation that the phantom limb was moving, enabling them to move their phantom limb into a more comfortable position (Figure 2.9). Praamstra et al. has established that this work induced more subtle expressions of motor cortical action during self-produced movements through a mirror [133].

Figure 2.9. Photograph showing mirror therapy being used in an upper limb rehabilitation program[134]

The focus of much of the work has been in rehabilitation in stroke patients [134-138], however, this technique is being used in the author's shoulder unit.

2.2 fMRI and Shoulder Movement

2.2.1 Overview

This will be a review of existing literature in relation to cortical representations related to motor function, their plasticity and some therapeutic interventions.

The intention is to focus on the literature of the upper limb that is relevant to the thesis, rather than giving a global overview of the following topics as they relate to the body as a whole.

2.2.2 Cortical Representation

The functional role of the brain within motor function has already been addressed in section 2.2. In this chapter a more detailed analysis will be undertaken of the telencephalon. Notwithstanding the impression that is created by reference to different areas/functions of the cortex, the components of the motor system

form a neural network, rather than isolated individual motor "centres", Figure 2.10.[139]

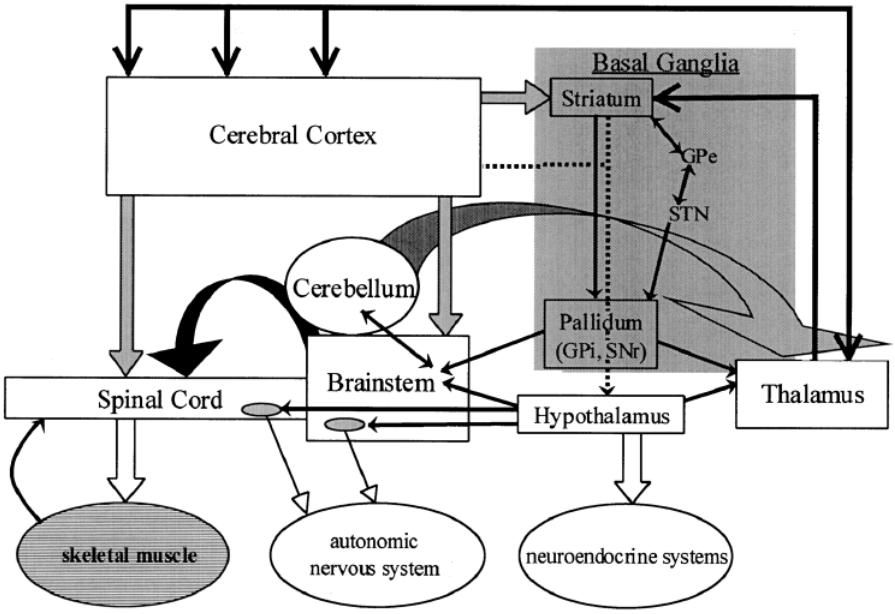


Figure 2.10, the major components of the motor system. The grey box represents the basal ganglia and the preganglionic autonomic motor nuclei are shown in ovals[139]

The Telencephalon is divided in the cerebral cortex and the basal ganglia. The motor function of the cerebral cortex can loosely be divided into the primary motor cortex (Figure 2.11, 2.12), the non-primary motor cortex and the prefrontal cortex. The main components of the Basal Ganglia [140], are the striatum, caudate nucleus and putamen.

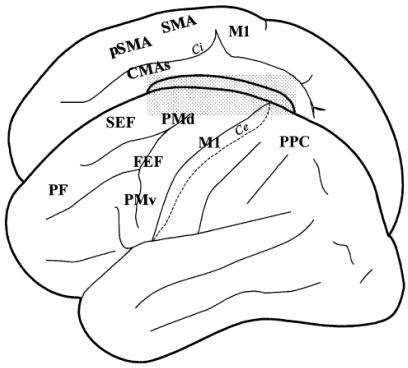


Figure 2.11 the motor areas of the human brain. The 'Dotted box' shows the - corpus callosum; Ce – central sulcus, CMA - cingulated motor areas, FEF – frontal eye field, M1 – primary motor cortex, PF – prefrontal cortex, PMd dorsal premotor cortex, PMv – ventral premotor cortex, PPC – posterior parietal cortex, SEF - supplementary eye field, pSMA presupplementary motor area, SMA – supplementary motor area.

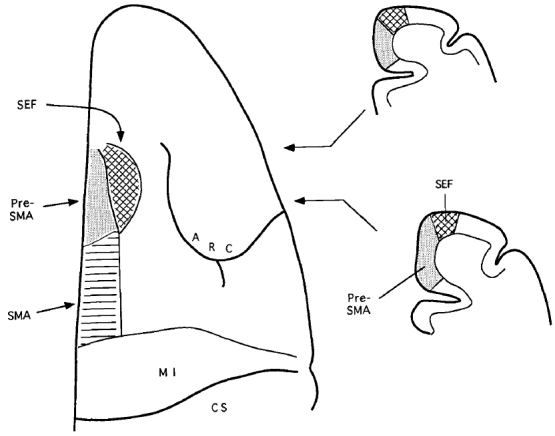


Figure 3, Schematic drawing showing the approximately locations of the Supplementary Motor Cortex, Supplementary Eye Field and the Pre-Supplementary Motor Cortex within the Frontal Cortex [141].

The cortical representation of the primary motor cortex can be traced back to the 1870s [142]. It was thought originally that control was manifest in well-ordered different cortical areas, controlling the face, arm and leg movements. These theories were conveyed in the iconic homunculus [143], (Figure 2.13). There has been a paradigm shift towards an understanding that cortical organisation is more complex. Further, the cortical representation of the face, arm and leg is more diffuse within their overall representation [144]. The cortical territory of individual muscles is more extensive and overlaps thus precluding spatially distinct territories for each muscle; [142, 145, 146]. Activation of a single muscle emanates from a number of locations within a section of the motor cortex, Figure 2.14. At an individual subject level the cortical organisation is more disfusely represented, in multiple rather than single areas alone, Figure 2.15. The interaoperative stimulation of the motor cortex which causes thumb movement could be provoked by three points along the central sulcus and simultaneously cause movement of others digits [141]. The level of activation related to the movement is also complex and not necessarily correlated to the complexity of the task or degree of movement involved and this is influenced by a variety of factors, such as the historical activity of the individual, with higher activation being exhibited in, for example, professional piano players [147].

Figure 4 – Internal and External Representation of Homunculus [142].

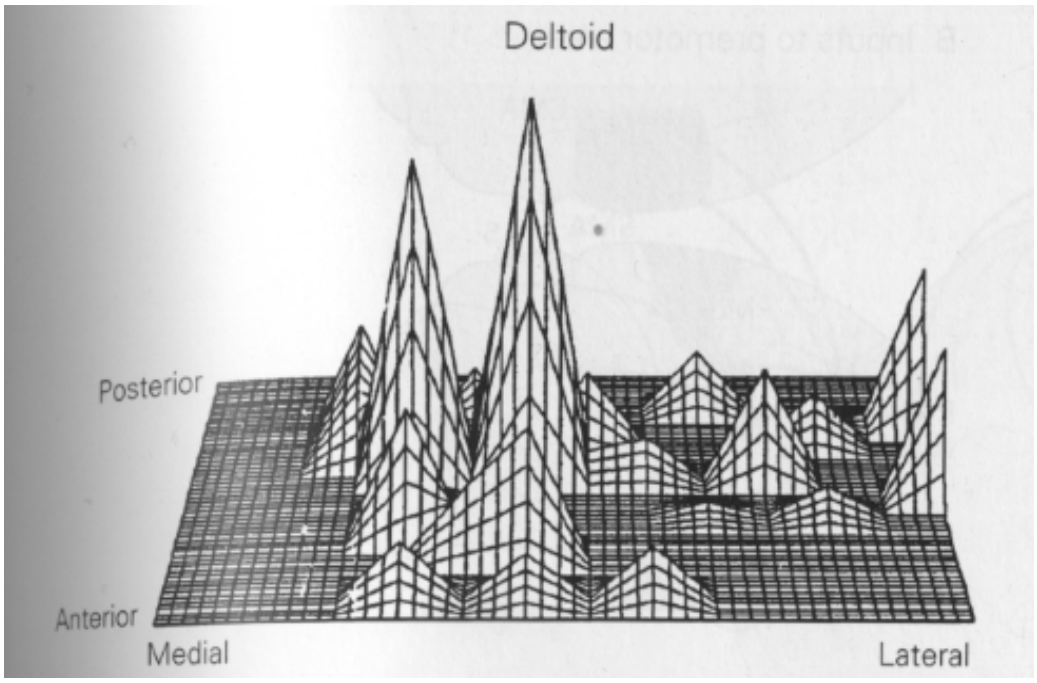


Figure 2.14 – Sites in the motor Cortex controlling the middle head of the deltoid muscle [148].

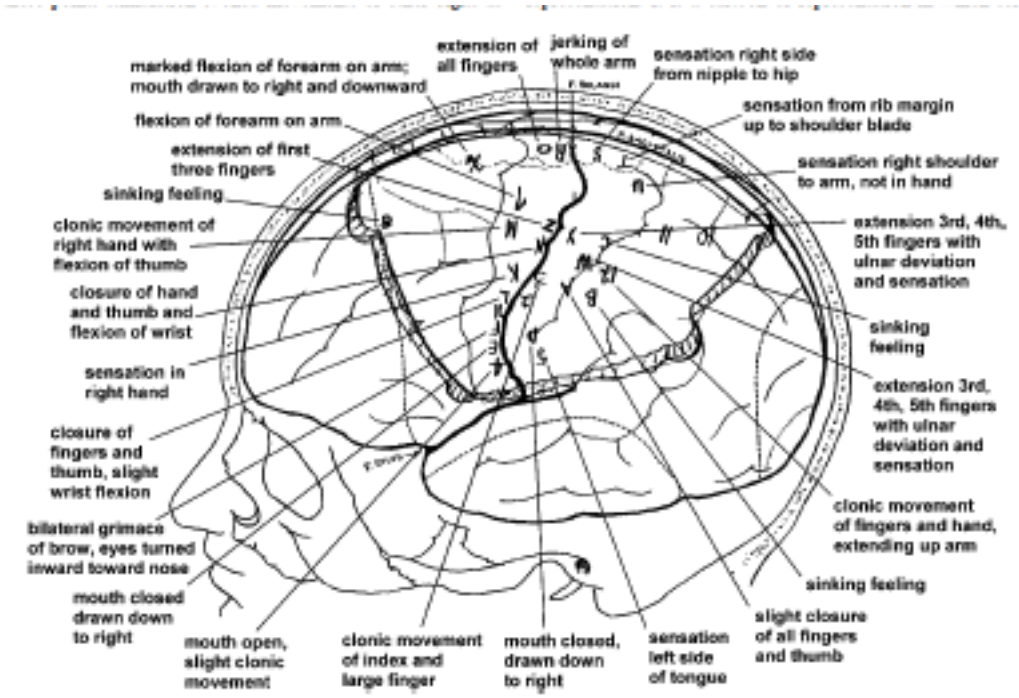


Figure 2.15 – Intraoperative stimulation of an individual human patient [148].

Endeavoring to establish the cytoarchitectonic of the cortex into functional distinct areas overwhelmed those undertaking the early work. But by the 1930s, work was undertaken on an animal model by Edgar Adrian, Wade Marshall and Philip Bard, which enabled Korbinian Brodmann to describe 52 physiological functional distinct areas. The work since this time has demonstrated greater numbers of functional physiologically distinct areas, many of the previously described areas containing discrete functional sub-areas [149].

The motor cortex is located in the dorsal aspect of the frontal lobe, Figure 2.16. It is involved in movement preparation [150], and motor execution particularly when the movement is more complex [141]. As well as the motor cortex having activations in a homunculus style, cortical representation activation is dependent on the stage of movement. Activation of the anterior-lateral region occurs in the preparatory phase and execution phase, whereas the posterior-medial region is only being activated during execution [150].

The motor cortex, Brodmann's Area 4, receives inputs from the following associated areas [151, 152]:

i. Pre-motor areas, Brodmann's area 6. The increased intensity of stimulation is required to provoke stimulation in this area feeding to the primary motor

cortex. This area tends to cause collective joint movements, produce complex movement rather than the primary motor cortex, which causes movement of a single joint. This can be further sub-divided into four areas:

a. Supplementary motor area – This has a rostral and caudal aspect; its role cannot be completely defined, but it is particularly important in precision movement [153] and it has a role in controlling postural centers [141].

b. Cingulated Motor Areas - This can further be divided into anterior and posterior areas. This is area is thought to be partially responsible for intiation of motor control, emotion, cognition, homeostatic drive, and reciprocal connections to the motor cortex; parietal cortex; limbic structures; brainstem nuclei; thalamus and spinal cord [154-156].

c. Lateral ventral premotor area – This area has the ability to stimulate bilaterally and is

consistent with a role of coordinated activity [157].

d. Medial ventral premotor area, is an area thought responsible for proactive inhibition of movement, prior to the execution of automatic sensorimotor processing [158].

ii. The Premotor area has strong interconnections between the above four areas, but in turn receives inputs directly and indirectly (Figure 2.16), from the following areas:

a. prefrontal cortex, directly from area 46;

b. basal ganglia, via ventrolateral nucleus, ventral posterolateral nucleus and nucleus X. These have a regulatory role over both the pre and primary motor cortex movement [159] and the preparation of action in [160];

c. Cerebellum. This area is responsible for the coordination of voluntary movement,

driving postural balance and co-ordination [153, 161].

d. The indirect connections of the basal ganglia and cerebellum, Figure 2.17 [150], are reciprocal, with each cortico-subcortical loop making a unique contribution to a particular motor behavior.

iii. Primary Somatosensory cortex, Brodmann's Areas 1,2 and 3 [162-164];

iv. Posterior Parietal areas, Brodmann's Area 5 and 7 - This area has an important role planning movement, containing a model of limb orientation[165] it is also thought to be proactive in the inhibition of movement [158].

A

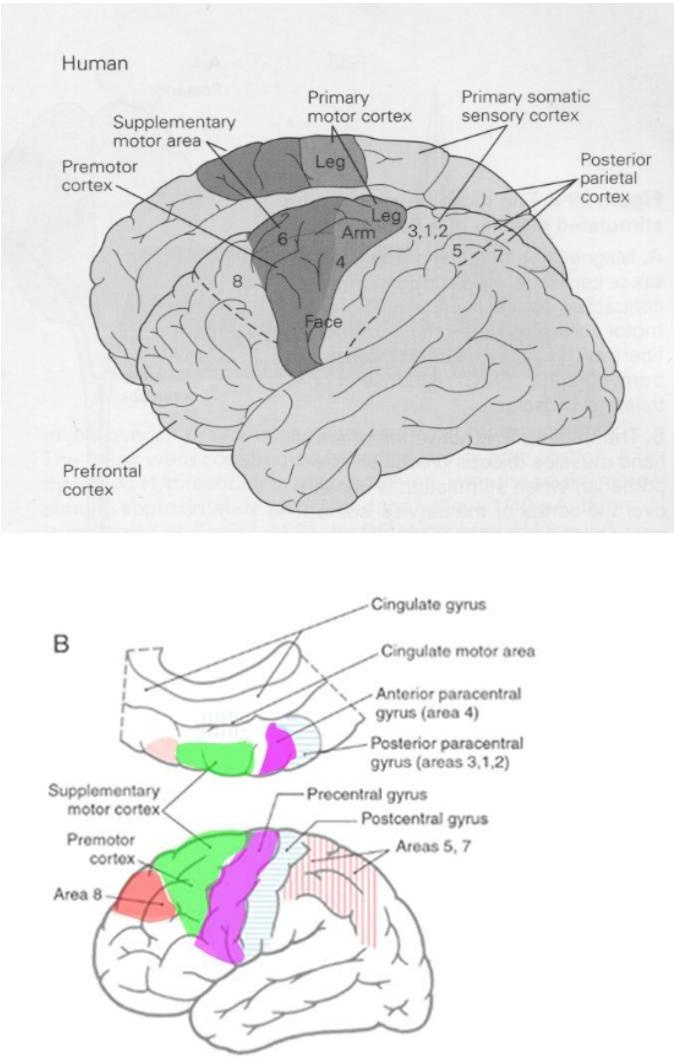


Figure 2.16 Cytoarchitecture of Motor Control:
A - Primary Motor Cortex and the Pre-Motor Cortex [151], B – Supplemental motor areas [141].

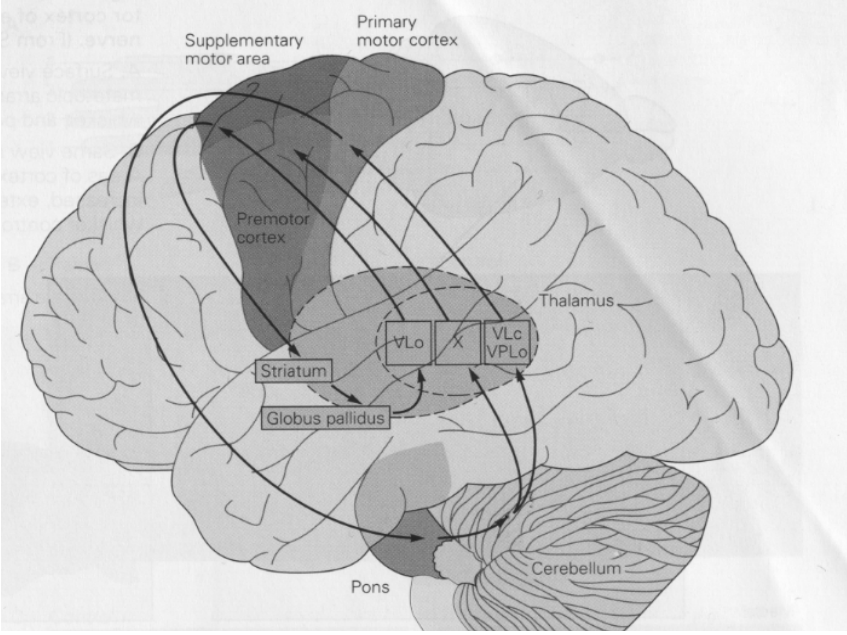


Figure 2.17 Indirect connection received by the primary and pre-motor cortex, VLo/VLc=rostral and caudal portions of the ventrolateral nucleus, VPLo=ventral posterolateral nucleus, X=nucleus X [150].

More recent studies performed on primates have shown that a single area of the motor cortex, perhaps even a single corticomotoneuronal cell, may have multiple functional connections to multiple muscles and multiple anatomical locations in the upper limb and thorax[166]. Thus, as well as there being at one level cortical representation corresponding to actions of the body, there are other levels with a complex network of functional neuronal connections which do not conform to simplistic homunculus representation, Figure 2.13)

Voluntary movement of the shoulder and other joints involves simultaneous contraction of multiple muscles, and this is reflected by a wide neuron activation in the motor cortex.[167] However,

counterintuitively the movement of a single finger provokes more cortical activation than multiple fingers. This is thought to reflect the need to stabilise the other parts of the upper limb to facilitate this movement [166]. This more diffuse cortical activation has been seen in other work. In a study of patients whom had hemiparesis and concurrent upper-limb hypertonus, the fMRI shows large amounts of bilateral activation in the sensorimotor areas, the supplementary motor areas and the cerebellum [168]. There is also suppressed excitability of the ipsilateral motor cortex during limb movement with the degree of inhibition dependent on the handedness of the individual. Further movement with the dominant hand invokes a greater ipsilateral suppression [169].

Reilly and Sirigu [170] found in their work with amputees that there are two levels of motor control, the motor cortex and a motor command map. They observed that following an amputation of either an upper or lower limb, the motor cortex is reorganised. However, they suggest that the integrity of the motor command map is maintained, causing the existence of the phantom limb. In upper limb amputees, imagining moving the phantom limb generated significantly higher activations in the contralateral primary motor and somatosensory cortex, compared to the controls. Further, in those patients with phantom limb pain, there was a cortical reorganisation in the motor cortex during imagined movements of the affected limb, which correlated to the amount of baseline pain they suffered [171].

The administration of anesthesia saw the rapid elimination of cortical re-organisation in these patients' somatosensory cortex, that was not seen in the patients with phantom limb pain who did not receive pain relieve [130].

This pattern of cortical re-organisation is different in those patients with peripheral nerve injuries, where the change in cortical activation is more complex. In these patients, although the upper limb cortical representation of the unaffected limb was maintained, there was an increase in level of activation. There was additional significant increase in activation of a number of premotor areas for unaffected upper limb movement, anterior cerebellum, supplementary motor, dorsal premotor cortex, post-central and parietal cortical areas. This change was more pronounced in the distal movement of the upper limb [172].

Specifically in respect of the shoulder in patients who have suffered a stroke, a greater cortical activation has been observed in shoulder movement of the affected side [173]. McKiernan et al also found that corticomotoneuronal connections possessed a greater comparative potency in the distal, compared to the proximal upper limb muscles [174]. This is consistent with lesions in the motor cortex which cause weakness in the distal part of the upper limb to a much greater extent compared to the proximal shoulder region [175]. However, the results in stroke patients needs to be treated with

caution as the majority of the work involves observations of lesions whose extent and location cannot be controlled experimentally or are conclusions drawn on animal models.

Cortical activation is further complicated by different types of unconscious movement that is generated after muscle contraction, known as involuntary movement [176]. Following contraction of a muscle after a latency period of between 2 to 7 seconds, there is an involuntary or unconscious further contract, which show a different cortical activation pattern compared to the initial contraction. The activation differences and similarities are apparent (Figure 2.18, Table 2.5); in particular in the involuntary movement there was greater activations in the anterior cingulated cortex (BA 24/32). This work demonstrates another complexity to the control of movement, but also serves to highlight the need for careful consideration of fMRI results.

Table 2.5, Significant functional brain activations present during voluntary movement [176].

Table 1 – Significant functional brain activations present during voluntary movement, involuntary movement (aftercontraction), and during the isometric 60 s MVC.

Condition	Cluster area (Gyral location)	Side L/R	Brodmann area	Z score (max)	MNI co-ordinates of max voxel
Right arm voluntary movement	V5/MT	R	19	6.10	46, −76, −6
		L	19	6.01	−46, −84, −6
	Caudate (head)	R		5.73	4, 18, 2
		L		2.52	−4, 18, 4
	Precentral gyrus	L	4	4.76	−24, −26, 58
	Superior temporal gyrus	R	41/42	4.55	40, 6, 2
		L	41/42	4.37	−46, 6, −2
	Superior parietal lobule	L	7	4.43	−38, −52, 62
	Cerebellum	R		4.39	10, −62, −18
	Thalamus	L		3.85	−12, −16, 22
	Putamen	L		3.59	−24, −14, 20
Right arm involuntary movement	Superior temporal gyrus	R	41/42	6.85	60, 8, 14
		L	41/42	3.82	−50, 4, 16
	Cerebellum	R		5.93	2, −66, −20
	Precentral gyrus	L	4	5.85	−32, −26, 48
	Superior parietal lobule	L	7	5.35	−38, −44, 68
	Anterior cingulate	R	24/32	5.31	8, 14, 38
	Thalamus	L		4.11	−18, −28, 16
	Medial frontal gyrus (SMA)	L	6	3.56	−4, −18, 54
	Caudate (head)	R		3.00	2, 14, 16
MVC of deltoid (isometric contraction)	Cerebellum	R		10.13	2, −68, −18
		L		6.80	−2, −70, −16
	Precentral gyrus	L	4	6.36	−26, −26, 56
	Thalamus	L		5.44	−4, −38, 2
	Caudate (tail)	R		5.29	24, −38, 16
	Superior temporal gyrus	R	41/42	4.27	60, 2, −2
	Superior parietal lobule	L	7	3.67	−38, −42, 66

All movements were performed with the right arm. Areas are ranked according to z scores.

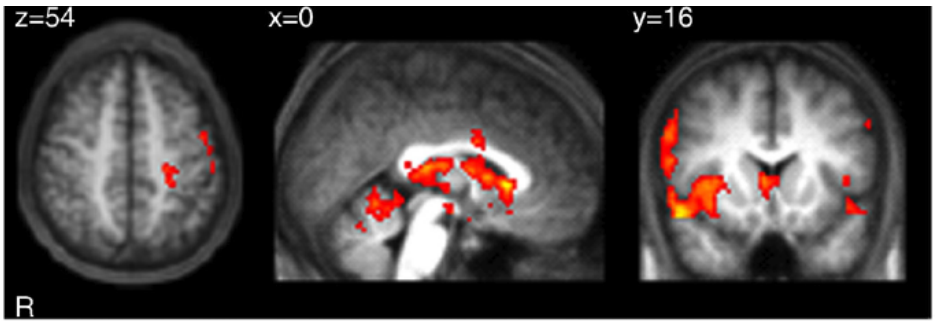


Figure 2.18, Brain regions showing a significant increase in BOLD signal (n=11) during voluntary movement of the shoulder [176].

Another factor that causes the cortical representation to be more complex is an individual's age. Although there is still controversy in this area, the motor tasks in the young appear to be on the contralateral side of the primary motor cortex, whereas cortical activation in the older shifts more to the ipsilateral side. The Supplementary motor cortex has a further variability; it is dependent on the type of activity, namely, activated in the young and not the old whilst undertaking upper limb movement, save when the movements are fine when both employ this region.[177]

The cortical activation is influenced by the type of movement, whether it is self-initiated or commanded. An fMRi study showed that in right finger movement, self-initiated movement produced comparatively larger activation in the Supplemental Motor Cortex; left parietal lobe; the left pre- and sensorimotor cortex and the right putamen [178]. Further, if the movement repeated and the individual trained in the movement, processing changes from the cerebellum and prefrontal cortex to the increased areas within the motor cortex which has been suggested, constitutes a site of long term memory of the acquired skill [179].

2.2.3 Lateralisation

The role of lateralisation or influence of “handedness”, has been the focus of a great deal of research attention [180], having a rich history dating back to Carl Wernicke in the 1870’s [181]. At a pure level it is unimanual movement instigated in motor cortex with the neural transmission to the contralateral hand via the corticospinal track [182].

The following is a summary of the main conclusions related to cortical representations:

i. Recent work in 2015 by Tzourio-Mazoyer et al. found that in 284 individuals lateralisation was not influential in motor activations [183]. This work is confirmatory of Hayashi et al.’s early work [184].

ii. Other work found that lateralisation caused increased bilateral activations and the extent of the motor cortex [185] [180] [182] [186].

iii. The nature of the movement has been found to affect the degree of lateralisation influence. Namely, repetitive and fine movements have been found to have greater influence on

laterally compared to the converse of these types of movement [184].

iv. In the tasks involving the hand, there is more bilateral activation when using the non-dominant hand;[185]

v. Lateralisation was influenced by the distal, as opposed to proximal movements of the upper limb. When shoulder movements where undertaken with the non-dominant hand the contralateral sensorimotor cortex activation was higher. Further, that shoulder movement activated by the sensorimotor cortex and the secondary areas, the activation of the primary areas being sparse in hand movements [180]

2.2.4 Neuroplasticity

The concept of plasticity of the motor cortex[187], or somatotopic re-organisation is not a modern discovery, Leyton and Sherrington in 1917[188], on the basis of their work on great apes, describe the concept as follows:

"….the functional instability of cortical motor points is indicative of the enormous wealth of mutual associations existing between separable motor cortical points, and those associations must be a

characteristically part of the machinery by which the synthetic powers of that cortex are made possible."

A more modern definition:

".. can be broadly defined as the ability of the nervous system to respond to intrinsic and extrinsic stimuli by reorganising its structure, function and connections; this can be described at many levels, from molecular to cellular systems of behavior and can occur during development, in response to the environment, in support of learning, in response to disease, or in relation to therapy". [189]

The difficulty is that monitoring cerebral activity of movement is complex; not only is there an interaction between motor and sensory component, but there is neural activation even when the limb is removed, so called "movement without movement".[190]

Although there have been advances in the understanding of neuroplasticity, some of which is described below, this has not translated into many established interventions [189]. Neuroplasticity can occur in response to injury/neurological pathology, neuroplastic-based interventions, neuropsychiatric disorders, development disorders, neurodegeneration and ageing.

One of the areas best-researched is neuroplastic change following a stroke [191], with changes to the location of cortical activation [192],

particularly in the motor and sensory cortex [193]; increased levels of activation in the contralateral cortical hemisphere [194-196] and changes in connections between network nodes [197]. Post-stroke the recovery in terms of adaptive cortical changes is influenced by many factors, including whether there in a neuroplastic intervention [198]. In Richards [199] et al. in a meta-analysis found that motor gains achieved through targeted rehabilitation were accompanied with neural changes in sensorimotor cortex lesion.

Neuropsychiatric disorders have an effect on neuroplasticity but rather than being caused by a lesion, they manifest themselves in individual alterations of the neural circuits in the limbic, prefrontal and frontostriatal with changes in motivation, perception, regulation of emotion[200], social ability, cognition and perception[189]. The pathogenesis is complicated by the neuropsychiatric disorders themselves, which are in turn influenced by polygenic risk factors such as substance use, psychological trauma, internal representation of self, social attachments and sociocultural influences [201]

It has long been known that paediatric patients recover to a greater extent following early neural insult, for example speech centres being found in the controlateral side following damage to the speech centre in the ipslateral hemisphere. The degree of plasticity has been demonstrated in congenitally deaf paediatric patients in their

response to cochlear implants; those under 6-7 [202] showing normal comparable cortical activation to non-deaf children, whereas those above this age demonstrate abnormal cortical activation.[203]

The ageing process has profound cortical effects such as changes in the cortical activation; cellular function; white matter integrity and brain volumes [204]. The cognitive reserve and plasticity have been suggested as an important compensatory mechanism for ageing effects [205]. The variability of both factors within the population, explains why individuals respond differently to the development of neurodegenerative diseases, such as Alzheimer's [204] and Multiple Sclerosis [206].

Work on neuroplastic-based interventions started in animal models. In rats plasticity has been demonstrated in motor skill acquisition [207]. After ten days of task training, microelectrode stimulation was employed to achieve high-resolution maps of the forelimb and hind limb representations of the motor cortex, Figure 2.19. The areas of increased representation reduced once the training ceased. A similar cortical expansion occurs in human subjects and is proportionate to the length of time for upper limb training [179], and has been established in lower limb training [208].

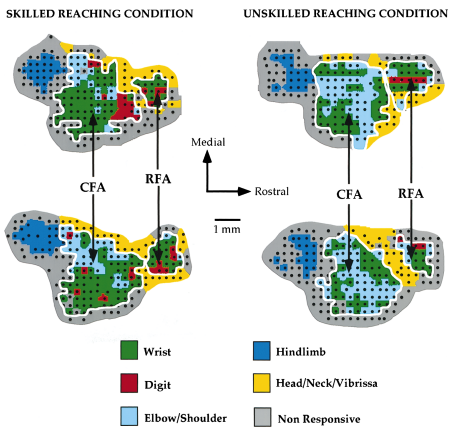


Figure 2.19 – Reorganisation of the Motor Cortex representation before and after skilled and unskilled training [179].

In human subjects, resting state fMRI has shown cerebral functional connectivity changes and increases in motor function for stroke patients following the use of a shoulder-elbow robotic rehabilitation paradigm [209], Figure 2.20. Neuroplasticity has been shown following stroke, where the cortical activation of the hand invades the shoulder region [192]. More recent studies employing motor imagery[1], executed movement and virtual reality-based training to attempt to use plasticity to overcome functional deficits following a stroke [210].

[1] Motor imagery is where an individual mentally rehearses the motor activity and

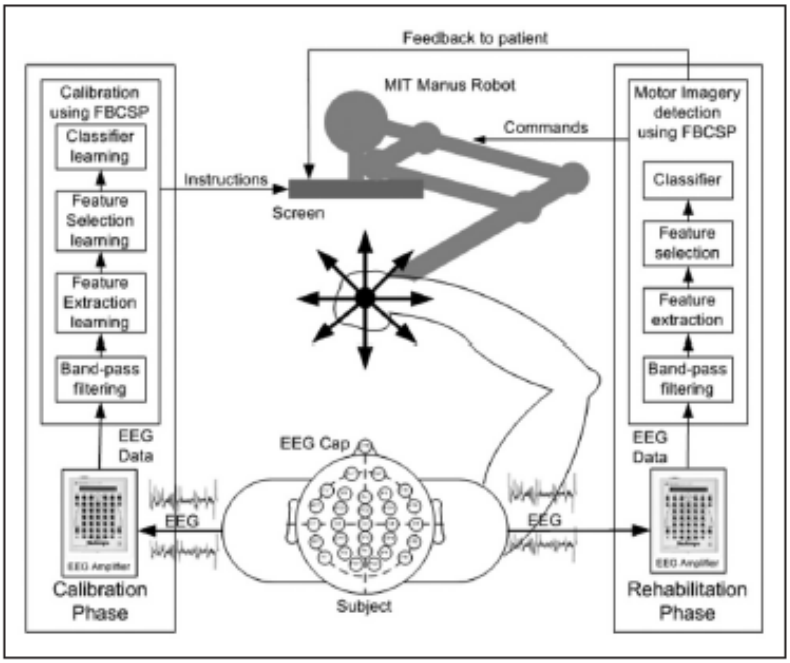


Figure 2.20 Architecture of MI-based brain-computer interface (MI-BCI) for upper-limb robotic rehabilitation (FBCSP, filter bank common spatial pattern; MI, motor imagery; BCI, brain-computer interface) [209].

Constraint-induced movement therapy ("CIMT") consists of restraining the unaffected limb and intensely using the effected limb, which has traditionally been used in patients who have suffered a stroke causing motor dysfunction. Improvement in stroke patients has been observed after such therapies, and fMRI has shown that other areas of the brain are important for the recovery of motor function, such as the ipsilateral cerebellum.[211] Even a short (12 day) programme of CIMT caused increase activation in the primary sensorimotor cortex in the affected hemisphere of the stroke patient [212]. The cortical changes as a result of CIMT in the various studies are not consistent, varying from recovery of the contralateral

sensory motor area of the effected limb to the increase in ipsilateral and bilateral activations of the sensory motor area [211].

2.3 EMG and Shoulder Movement

The following section aims to provide a brief overview of EMG shoulder research in both the normal and abnormal shoulder.

2.3.1 Introduction

The efficiency of EMG as a direct measure of muscle activity has been well established, with the muscle activity measured through mfMRI being directly correlated with EMG activity. This correlation was originally established by Adams et al. [213], and subsequently validated by others such as Kinugasa and Akima [214].

To effect purposeful movement in a normal shoulder, there has to be synergism between muscles, neural connections, cortex and behavior. Early EMG shoulder research focused on the activities and injuries of professional and amateur baseball players [215] and others pursuing different sporting activities [216]. Advancement of understanding has to a certain degree been hampered by conflicting results and a lack of confidence in the methodology adopted. Recent work has identified the need to undertake EMG studies of

simple movements in order to establish a solid base of reliable knowledge [217, 218]. Forward flexion and abduction are simple movements but they underlie all activities of daily living. A thorough elucidation of the simple movements [219] through EMG [220] is critical before a better understanding of more functional tasks [221] can be achieved.

It is important to realise that, in order to study shoulder movement and thus skeletal stabilization, we need to consider muscles not traditionally thought involved in shoulder movement such as transversus abdominis [222], lumbar multifidus [223] and gluteus maximus [224].

When studying shoulder movement and muscle activity, there is a great deal of silent activity prior to movement of the upper limb. This includes anticipatory postural adjustment, proprioception, muscle activity before movement, centre of gravity stabilisation, and effects of mental state, fatigue, pain and posture [225-227].

The activity of pectoralis major and latissimus dorsi, which are believed to have a pivotal role in shoulder stabilisation, are often neglected in EMG studies of shoulder movement and instability [218].

2.3.2 EMG in Normal Shoulder Movement

The muscles in the shoulder have a critical role in maintaining the humeral head in the central glenoid which maintains joint stability [228] and is achieved by muscles acting in a synergistic manner. So in forward flexion, muscles of the anterior rotator cuff are balanced by those in the posterior rotator cuff, which prevents humeral head translation caused by torque-producing shoulder muscle movement [217].

The clinical importance for examination of the shoulder and the surrounding joints has long been recognised. In 1944, in Inman's landmark article [229, 230] simple elevation of the arm either through forward flexion or abduction was thought to expose a great deal of pathology. Cumulatively, 60 subjects make up the sum of EMG knowledge in simple movements for forward flexion and abduction/adduction in the major studies of Krongberg et al., Apert et al., David et al., and Wickham et al. [231].

Interestingly, the history of the individual activity can alter the activations of shoulder muscles. During pitching, professional baseball players activate supraspinatus and latissimus dorsi, whereas amateurs demonstrate increased activity of supraspinatus and infraspinatus [232].

The body position of the individual greatly influences the muscle and the level of activation, and this can be readily demonstrated in EMG work undertaken in extension and flexion in the prone position (Figure 2.21). Subtle effects of undertaking rehabilitation exercises lying on the side have also been found [219, 233], but little work has considered muscle activation in the supine position. It can be seen that latissimus dorsi has one of the highest levels of activation, whereas in a standing position this movements would generate high levels of activation in anterior, middle and posterior deltoid.

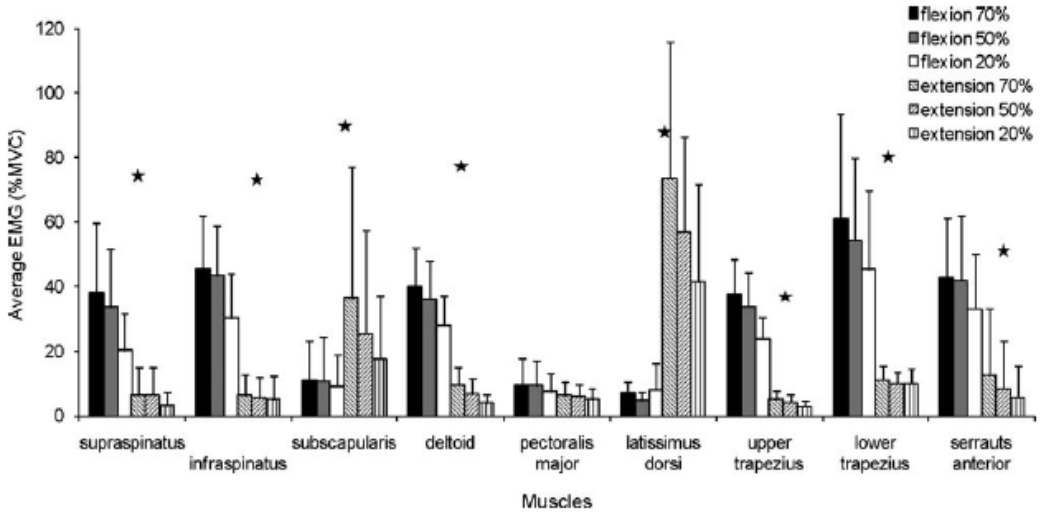


*Figure 2.21 – This graph to shows the EMG signals (%MVC) from nine muscles during flexion and extension in the prone position at 20%, 50% and 70 of maximal load. The * symbol indicates the difference in muscle activation between flexion and extension [217].*

2.3.2.1 Shoulder Flexion

Shoulder flexion is commonly examined in clinical practice and although a simple movement, there are still gaps in the understanding of the muscle patterning which enables it [234]. A

large number of muscles are involved in forward flexion of the shoulder, and their level of activation, timing and interrelationship are complex.

It can be seen that maximal voluntary contraction in forward flexion has to balance muscles that action on opposition to stabilise the humeral head on the glenoid. Strong activation of anterior deltoid to achieve the elevation is counter-acted by serratus anterior, subscapularis and teres minor, Figure 2.22.

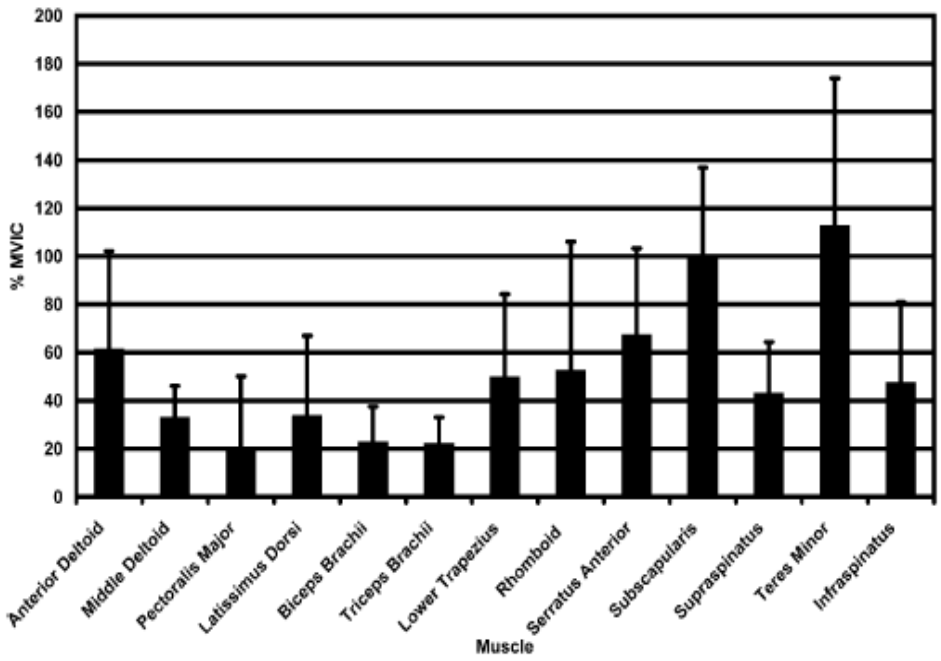


Figure 2.22 – This graph shows the maximal voluntary contraction of a number of shoulder muscles during the movement of forward flexion [215].

Heuberer et al. shows the muscle activation changes for 14 muscles over the range of motion of forward flexion (Figure 2.23). This muscle patterning is essential to maintain the stability of the shoulder. For example, teres minor has a greater action after 90 degrees in forward flexion; this causes posterior depression at the glenohumeral joint, maintaining stability.

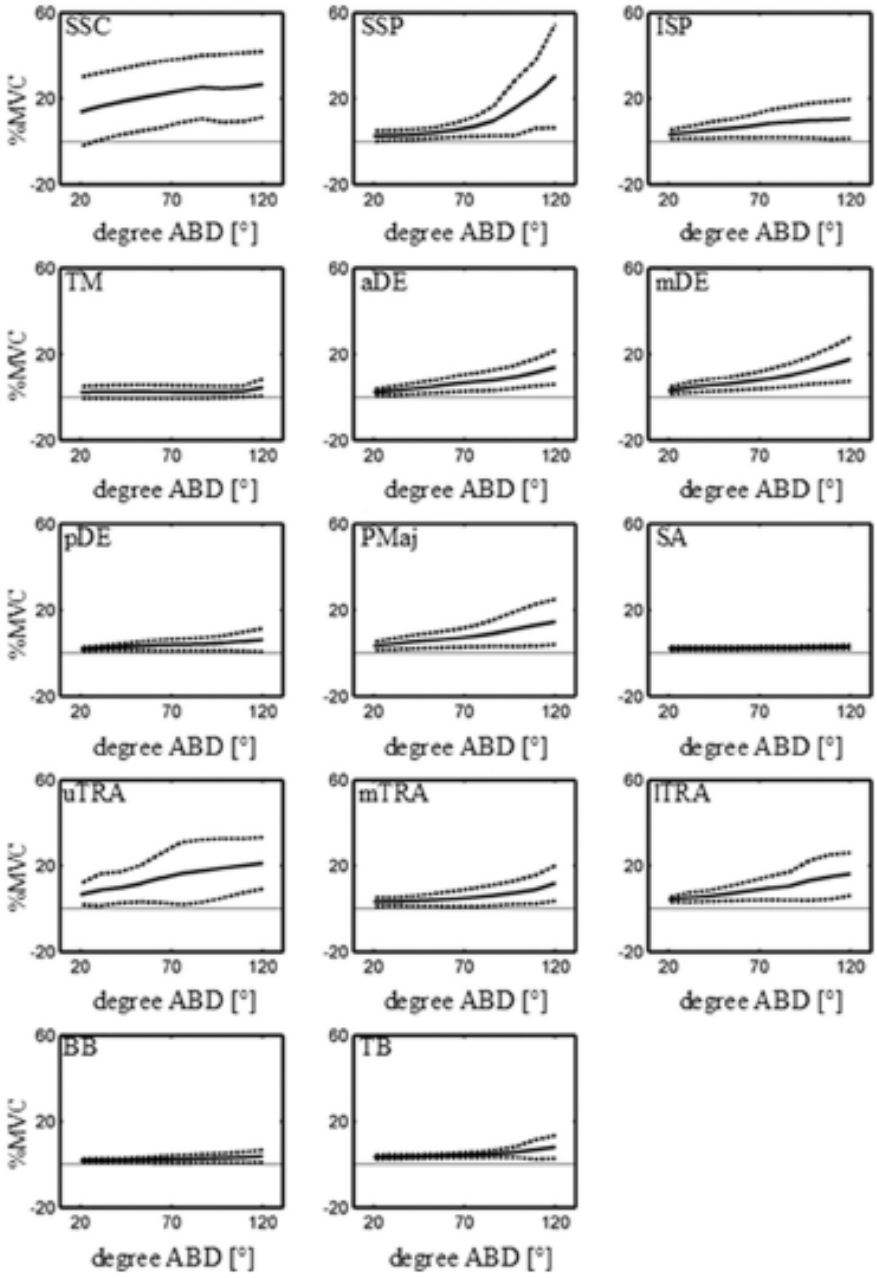


Figure 2.23 – The graphs show normalised activity of 14 muscles during concentric forward flexion. The middle line represents the mean intensity of signal, expressed as a percentage of the whole concentric movement. (SSC is subscapularis, SSP is suprascapularis, ISP is infrascapularis, TM is teres minor, aDE is anterior deltoid, mDE is middle deltoid, pDE is posterior deltoid, PMaj is pectoralis major, SA is serratus anterior, uTRA upper trapezius, mTRA middle trapezius lTRA is lower trapezius) [218].

The only study that considered individuals in a supine position relates to shoulder rehabilitation exercise [235] . The conclusion is

that muscle activity increases with more demanding shoulder rehabilitation exercises, namely from passive to active movement. However, It does not consider the muscle patterning in simple shoulder movements such as forward flexion or lateral abduction. The EMG research undertaken has the patients either in a standing or sitting position [234, 236]. There is therefore a gap in knowledge, and so assumptions are made that there is no material difference in muscle activation in the shoulder in a supine position compared to standing. This is particular relevant in fMRI which can only be undertaken in the supine position.

We can distill the following from the literature, for shoulder flexion in an individual in either the standing position or vertical in the sitting position:

i. The posterior cuff muscles act synergistically to counteract the translational forces generated in forward flexion [234].

ii. Townsend et al., confirmed previous EMG studies that found anterior and middle deltoid muscle were important in coronal, sagittal and scapular planes in forward flexion [237].

iii. Some work has found that subscapularis has a high level of activity in concentric forward flexion and abduction [218].

2.3.2.2 Shoulder abduction/adduction

Abduction is mainly a result of the anterior, middle and posterior deltoid muscle (Figure 2.24). As the arm abducts, the humeral head shears against the glenoid, and superior-inferior translation has been estimated at 2.6 mm [238].

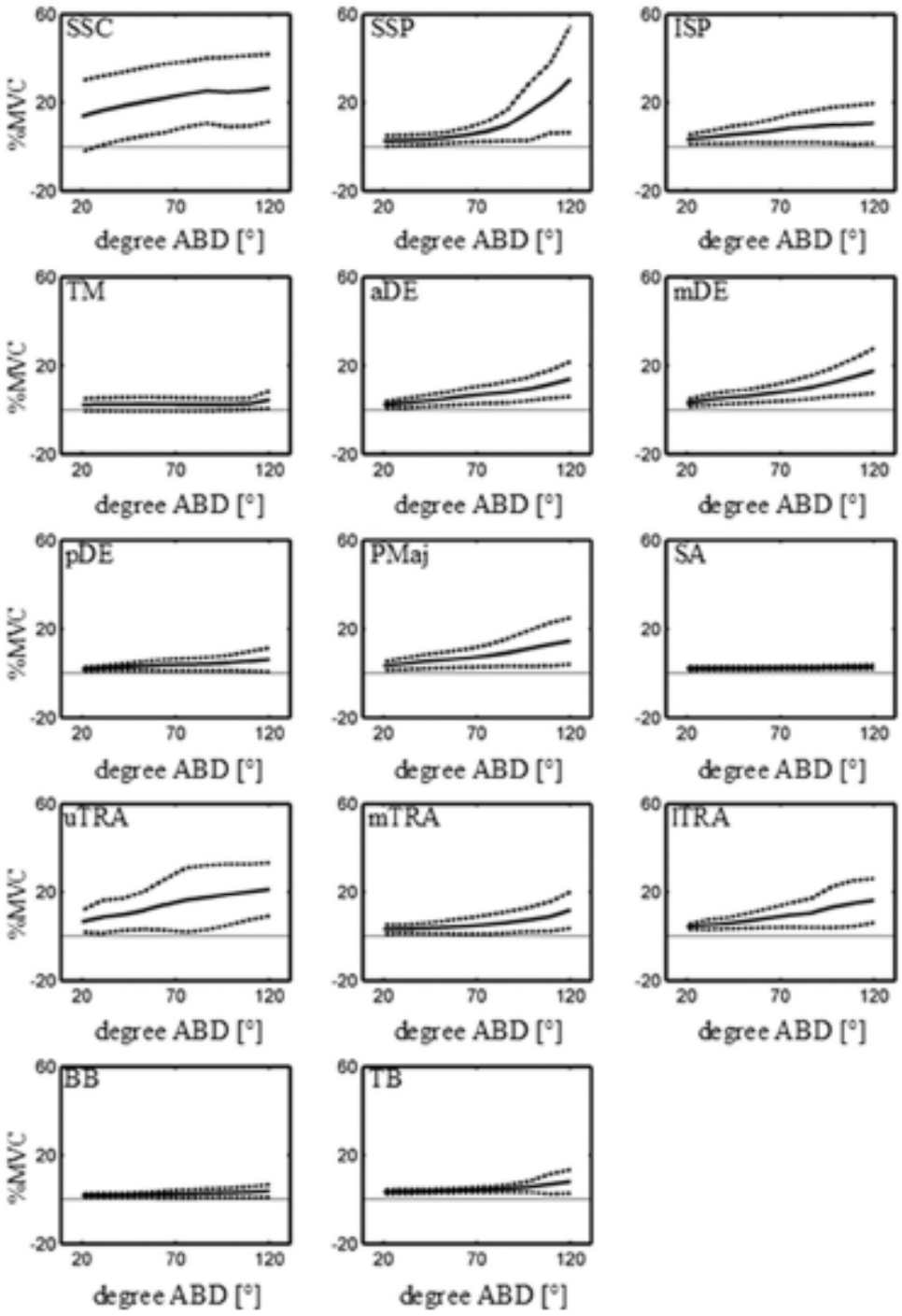


Figure 2.24 – The graphs show normalised activity of 14 muscles during concentric abduction. The middle line represents the mean intensity of signal, expressed as a percentage of the whole concentric movement. (SSC is subscapularist, SSP is suprascapularis, ISP is infrascapularis, TM is teres minor, aDE is anterior deltoid, mDE is middle deltoid, pDE is posterior deltoid, PMaj is pectoralis major, SA is serratus anterior, uTRA upper trapezius, mTRA middle trapezius lTRA is lower trapezius) [218].

We can distill the following from the literature for shoulder abduction/adduction for an individual in either the standing position or vertical in the sitting position:

i. Anterior deltoid, middle deltoid, infraspinatus, supraspinatus are involved in abduction [239].

ii. Subscapularis is active at the beginning of a concentric movement, and gradually increases as the angle increases [218].

iii. As expected, Heuberer et al. demonstrates that the EMG activations for anterior, middle and posterior deltoid are the same in this movement, and there is a correlation with pectoralis major [218].

iv. Serratus anterior and biceps brachii in this movement show no significant activity, although the activity of serratus anterior is greater in abduction compared to forward flexion, which is related to the increased need to stabilise the scapular on the trunk [218].

v. Some work has found that subrascapularis has a high level of activity in both concentric abduction and forward flexion [218].

vi. As the angle of abduction increases, subscapularis and infraspinatus activity increases in an antagonistic role to stablise the glenohumeral joint against gravity [218].

2.3.2.3 Pectoralis Major and Latissimus Dorsi

The co-contraction of these muscles has been shown to be pivotal in preserving glenohumeral stability with shoulder cuff pathology [240].

Historically pectoralis major and latissimus dorsi tended to be ignored in studies of central muscles involved in shoulder movement [241]. An EMG study some time ago, demonstrated that pectoralis major was inactive during abduction [239, 242] however, recent work has suggested that this increases during abduction which indicates that it acts as an agonist in this movement [215, 218]. Under-activation of latissimus dorsi and pectoralis major is thought to correlate with shoulder instability, and in particular an imbalance of rotator cuff co-activation [215].

Glousman et al. [243] found in professional baseball players with shoulder instability, a marked reduction in the activity of both pectoralis major and latissimus dorsi whilst pitching. The conclusion was that this reduction in the muscles activation increased instability, by causing decreased internal-rotation forces that are required during the later part of the throwing technique.

In multi-directional instability, it can be seen (Table 2.6) that there are significant differences between the activation of pectoralis major in patients compared to the controls. However, further comparison of latissimus dorsi between the two groups showed no difference [244].

Table 2.6 – Table to show the EMG signals in latissimus dorsi and pectoralis major of patients' with multi-directional instability compared to controls (AMP(%)=normalised peak EMG,(% peak EMG), ONST=onset of activation, TERM=termination of activation, ROM=activation range of motion) [244].

Control and MDI group mean (SD) EMG parameters for the latissimus dorsi and pectoralis major

		Latissimus dorsi				Pectoralis major			
		AMP (%)	ONST (°)	TERM (°)	ROM (°)	AMP (%)	ONST (°)	TERM (°)	ROM (°)
Abduction	Control	49.7 (15.7)	2.7 (8.5)	−40.1 (49.8)	127.4 (50.7)	–	–	–	–
	MDI	59.2 (22.9)	7.2 (18.8)	−9.7 (59.6)	92.5 (57.5)	–	–	–	–
Adduction	Control	51.8 (13.0)	−8.5 (9.1)	21.7 (28.7)	76.9 (28.3)	51.3 (11.7)	−5.7 (4.2)	21.1 (18.9)	74.6 (20.7)
	MDI	52.6 (10.8)	−7.6 (8.3)	26.0 (24.5)	72.0 (26.4)	46.6 (11.4)	−5.3 (8.5)	18.3 (45.8)	76.9 (50.1)
Flexion	Control	53.1 (11.5)	−5.0 (11.2)	−18.5 (28.2)	113.4 (34.4)	53.7 (16.7)	−7.4 (10.5)	13.0 (58.3)	84.4 (62.3)
	MDI	52.9 (11.0)	−6.9 (12.1)	−20.2 (27.3)	117.1 (34.5)	50.5 (20.8)	−10.6 (11.2)	−2.0 (53.6)	102.6 (59.8)
Extension	Control	–	–	–	–	62.6 (16.2)*	−2.0 (1.8)	32.9 (41.7)†	59.6 (41.6)†
	MDI	–	–	–	–	37.3 (8.0)	−5.2 (3.8)	−29.2 (38.8)	124.4 (40.3)
Internal rotation	Control	59.6 (8.0)	0.0 (1.9)	−0.1 (38.1)	90.1 (37.8)	46.6 (12.6)	−3.7 (2.9)	23.1 (36.5)	70.6 (37.7)
	MDI	55.8 (22.2)	−0.2 (5.1)	−18.3 (65.0)	107.7 (62.9)	45.3 (7.6)	−4.7 (5.2)	17.8 (18.4)	76.9 (20.5)
External rotation	Control	60.0 (12.4)	−4.8 (21.0)	7.5 (20.9)	87.3 (29.6)	–	–	–	–
	MDI	57.0 (9.9)	−16.9 (25.5)	−4.9 (34.2)	111.8 (45.4)	–	–	–	–

The * indicates a significant difference of $P<0.005$ between the control and MDI groups, while the † indicates a difference of $P<0.01$.

The structure of pectoralis major is that there are two heads, clavicular and sternocostal. The activation of these two heads is dependent on the inclination of the thorax. The focus of the EMG

work has been during complex activities rather than simple movements, which may be assessed clinically [245, 246].

There has been little research into latissimus dorsi anatomical variation until recently. Anatomical variation of latissimus dorsi was explored by Pouliart and Gageys' [89] in 2005, based on the study of 100 cadaver specimens. They found latissimus dorsi tendon insertion on the humeral head was variable (26.06 mm +/- 5.11mm) and 43% had muscular fibers arising from the inferior angle of the scapula. This morphological variation needs to be taken into account when evaluating the result of EMG and other modalities. Pouliart and Gagey's further papers were based on this cadaveric work, which advanced the theory that latissimus dorsi, and in particular its tendon footprint location, may be implicated in spontaneous reduction of dislocation and instability generally [247].

In order to maintain the humeral head centre over the glenoid, to limit translation, muscles around the shoulder act as couples in an agonist and antagonist role. Latissimus dorsi along with teres major acts in an antagonist role to deltoid isometric and dynamic concentric elevation [248].

Horsley et al, based on their work with professional rugby players, thought that over-activation of latissimus dorsi caused a compensatory mechanism of abnormal muscle patterning. This

dysfunctional muscle patterning then caused the players to have unstable shoulders [216].

2.3.2.4 Other muscles involved in shoulder movement

In EMG studies of forward flexion and adduction/abduction, trapezius and serratus anterior has shown the greatest activity during the upper end of concentric and end of eccentric movement [249]. McMahon et al. found that serratus anterior and supraspinatus under-activation correlated with shoulder instability [250]. This is an important finding, but this work only studied supraspinatus, infraspinatus, subscapularis, serratus anterior, rhomboids and trapezius. Other studies have not found serratus anterior to be critical in shoulder movement [216].

Supraspinatus and infraspinatus are activated in seemingly minor tasks where coupling across the joint would seem unnecessary: for example, passive forward flexion of the shoulder in a table-sliding exercise undertaken following arthroscopic repair of the rotator cuff (Figure 2.25) [70].

Figure 2.25 – Photograph showing the table sliding exercise that is undertaken at some centers following an arthroscopic repair of the rotator cuff [70].

Muscle activation is dependent on the type of movement (Figure 2.26), however, specific patterns can be recognised in EMG studies of simple shoulder movements in specific angles. Sakai et al. studied the patterns of infraspinatus, anterior deltoid, middle deltoid and supraspinatus [219]. Supraspinatus was more dependent on the angle of movement in the transverse plane (Figure 2.27) and tended to have a reverse patterning to Infraspinatus.

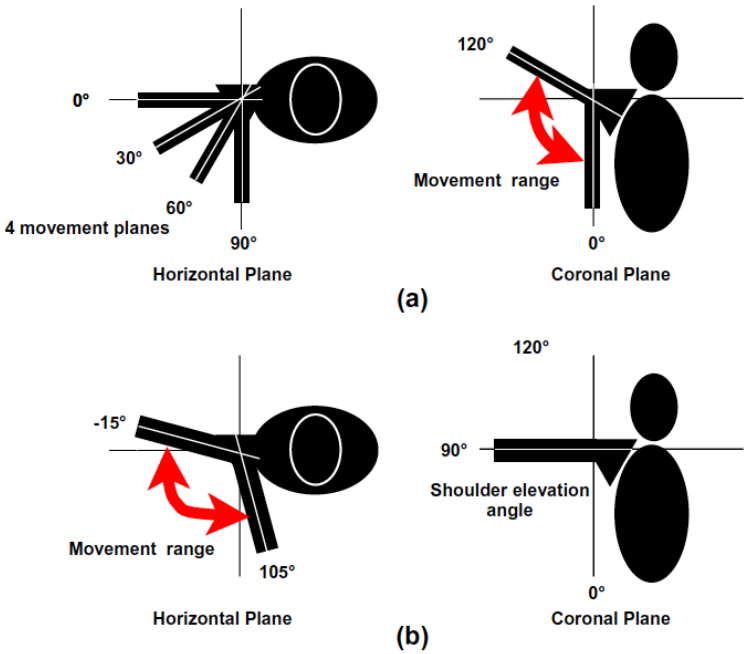


Figure 2.26 – Diagram to show the shoulder movements during EMG in the study of Sakaki et al. [219].

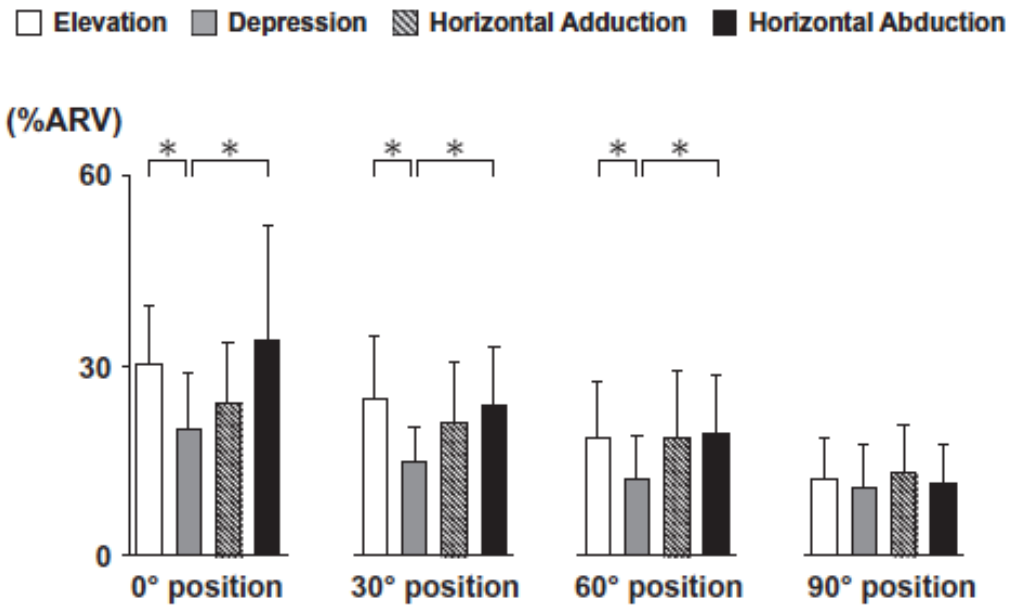


Figure 2.27 – Graphs show the percentage average rectified values, for supraspinatus in for the movements of elevation, depression and horizontal abduction/adduction [219].

2.3.3 EMG in an Abnormal Shoulder Movement

Abnormal or dysfunctional shoulder movements are due to multiple factors. In patients with stroke, for examples, contributing factors are abnormal cortical muscle representations [251], abnormal recruitment of agonist/antagonist muscles [252], muscle hypersensitivity/weakness [253, 254], impaired sensation, spasticity and weak functional coupling [236, 255]. EMG has demonstrated a reduced synergy between anterior deltoid and triceps compared to controls, with a correlation to reduced function in stroke patients [236].

In EMG studies of a small sample of patients with generalised laxity (6 patients and 5 controls) supraspinatus and subscapularis showed greater activation than the controls. The hypothesis advanced was that this additional activity was to provide anterior shoulder stability in the lax joint. Further, the work was comparable with previous studies that considered concentric and eccentric movements in this patient group. The patients with shoulder laxity had significantly lower muscle activation during eccentric movements compared to controls [239].

Barden et al. studied patients with MDI [244] and drew several conclusions, based on comparing a small number of patients (7 patients):

i. That the muscle patterning of the shoulder muscles studied were different between the two groups.

ii. Posterior deltoid, infraspinatus and supraspinatus in the MDI group showed shorter periods of activation and different timing of activation although there was no difference in levels of activation between the two groups. The posterior deltoid had a delayed onset of action in certain movements, internal rotation, which may compromise the anterior stability of the shoulder.

iii. The activation of pectoralis major was significantly different in this group, maintaining a low constant activation in forward flexion, abduction, internal/external rotation rather than distinctive patterns of activation and deactivation.

iv. That MDI was caused by a neuromuscular control deficiency that affects the coordination of the rotator cuff.

As mentioned in the literature review that relates to shoulder instability, Chapter 2.1, the definition of shoulder instability is problematic, with studies often treating a patient group as homogenous despite there being mixed causative pathologies. This is particularly an issue in the studies of Barden et al. [244], and Moseley et al. [241], where patient groups are not clearly defined.

Hawkes et al. [248] showed a compensatory stabilisation in patients with massive rotator cuff tears by activation of rotator cuff muscles, subscapularis, infraspinatus and supraspinatus in concentric forward flexion. In the same patient group there was increased activity also in middle deltoid, pectoralis major, serratus anterior and upper trapezium; that was thought to be a compensatory mechanism to maintain stability [248].

Steenbrink et al. [256], has established through inverse dynamic simulation modeling that rotator cuff tears beyond supraspinatus lead to instability. Muscle activation changes as deltoid in an attempt to retain abduction torque, introduces destablising forces, with subscapularis, teres minor and bicep muscles to maintain the abduction torque and glenohumeral stability [256].

3 Methodology

In this chapter I describe the methods used in the pilot study, the reproducibility study and then the main study protocol.

3.1 fMRI and Motion Capture Method Development Study

3.1.1 Introduction

The fMRI techniques are set out in Chapter 1.4.2. It was important to establish that the proposed movement protocol produced valid and reproducible results. I was also keen to establish the exact extent of the movement in the scanner, which could be achieved by using motion capture.

3.1.2 Objectives

The objectives of the method development study were as follows:

1. To measure the upper limb movement using motion capture to assess the range of motion in the scanner.

2. To ensure that this setup generated technically meaningful data.
3. To assess the practicalities of the proposed shoulder movements in the MRI scanner.
4. To identify any technical issues with the equipment.
5. To confirm that cortical activations were seen in the appropriate area in normal subjects.

3.1.3 Participants

All the participants were university students or members of the university staff (Table 3.1), who had no history of upper extremity pathology. Prior to attendance at the data collection session, the participants had been provided with a Patient Information Sheet, Appendix 1. Before the session commenced the participants had the opportunity to ask any questions, after which they signed a consent form if willing to proceed, Appendix 2.

During the method development, movement of the right upper limb was undertaken.

Table 3.1. The demographics of the participants in the fMRI pilot study (n=4)

Age	30.8 (20-41) years
Sex	2 Male / 2 Female
Handedness	0 Left / 4 Right

3.1.4 Method

3.1.4.1 fMRI Study

Chapter 1 gives the background to the methods used for testing, signal processing and data processing.

The participants undertook 10 cycles of movement, consisting of two types of movement and two rest periods (Figure 3.1, 3.2), each of 12 second duration. The order of movement tasks between forward flexion and abduction was randomised. Other studies had found a difference between unpredictable tasks and predictable tasks in finger movements [257]. Had the task been a predictable movement order, namely, a regular pattern of forward flexion/abduction (repeated), there would have been a risk of introducing a

confounding factor as the type of cortical motor activation would change from the initial unpredictable to predictable.

The upper limb through shoulder joint has in itself 6 degrees of freedom, forward flexion, extension, abduction, adduction, rotation and circumduction. It was important to try and standardise the cortical activation produced by the shoulder movement. In order to reduce inter-subject variability in the pathway of upper limb movement, prior to scanning the subjects were shown the frequency of movement (1 Hz) and shown how to lock their elbow and wrist. The type of movement was communicated to the participants by projecting colored light onto the scanner, which was visible from the mirror within the head coil.

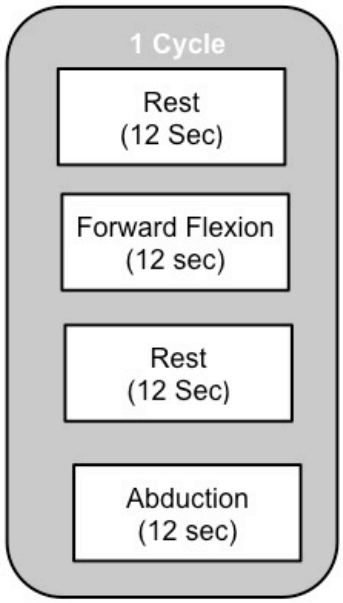
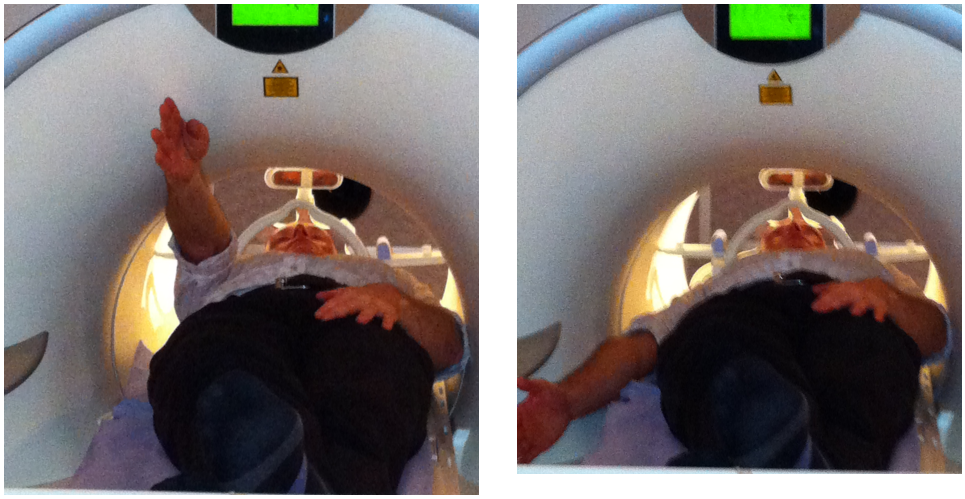


Figure 3.1. Diagram illustrating one of the 10 cycles that was undertaken in the scanner. The order of the movement was randomised.



A B

Figure 3.2. Photographs showing the movement of forward flexion (A) and abduction (B) in the Siemens 1.5 T Scanner

The data was processed using SPM 12 (University College London), [258-263], as set out in Chapter 1.4.2.1. At the first level the 6 movement parameters were modeled as multiple regressions to reduce the confounding effect of the movement. The model was then estimated. Two contrasts were computed at the first level, with rest subtracted from forward flexion and abduction. The same pre-processing and first level model steps were adopted for the pilot study as in the main study. As the second level a 2 x 2 factorial model (Figure 3.3), the first created for forward flexion and the second level created for abduction.

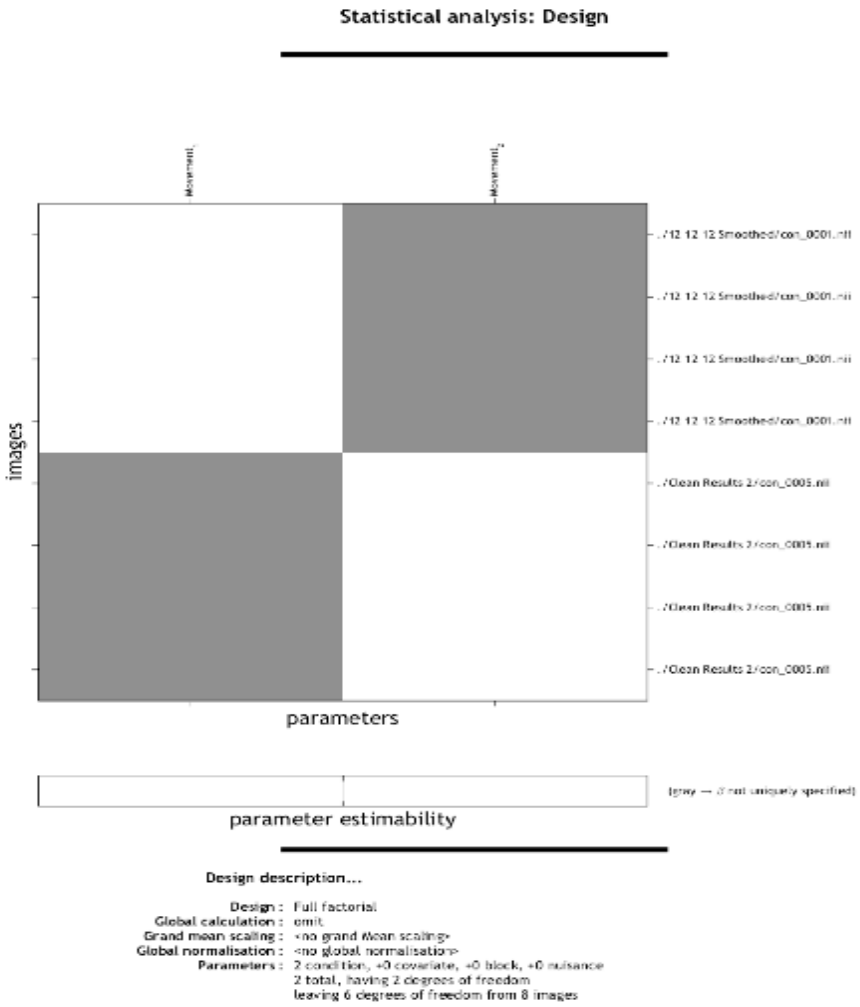


Figure 3.3. Second Level factorial model generated in SPM12, the first level is all movement and the second level is abduction subtracted from forward flexion

3.1.4.2 Motion Capture Study

The purpose of this part of the study was to calculate the exact movement of the upper limb in the 1.5 T Siemens scanner (Figure 3.4 photograph A). Due to the powerful magnetic field of the scanner, it was critical that the (non-magnet-safe) equipment was kept at a safe distance from the scanner. A safe zone around the scanner was defined with reference to the scanner specifications and the research team was briefed.

The participants in the scanner undertook the movement as set out in the previous section, whilst the motion capture cameras recorded the position of the reflective markers. Prior to the participant being placed into the scanner, a static recording was made of the individual (Figure 3.4, photograph B). From this static trial a model could be estimated, which enabled the position of the upper limb to be estimated when only the distal markers were visible (Figure 3.5).

A B

Figure 3.4. Photograph showing motion capture study. Photograph A shows the motion capture cameras and the Siemens MRI scanner. Photograph B demonstrates the upper limb with the reflective markers used to create the upper limb model.

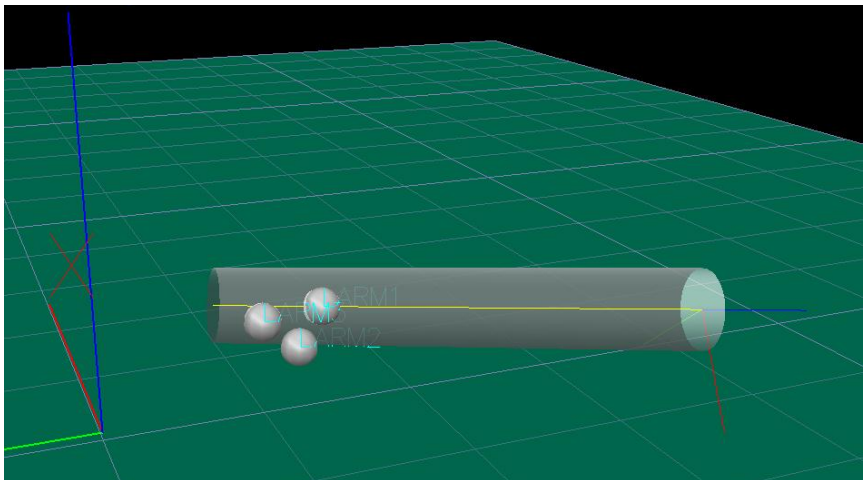
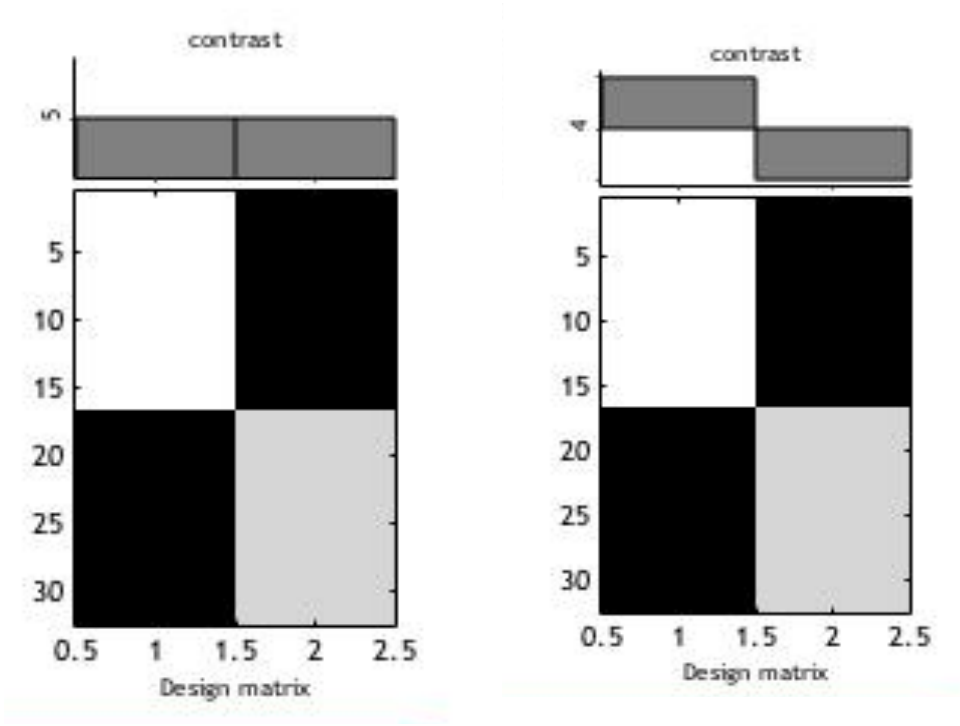


Figure 3.5. Screen image from Visual 3D, Sweden, showing the upper limb model that was created to estimate the upper limb movement whilst in the scanner.

3.1.5 Results

3.1.5.1 fMRI Study

Figure 3.6 illustrates the 2 x 2 factorial model designs, with the two contrasts that were relevant. The first matrix (Figure 3.6. A) shows forward flexion and abduction. The second matrix (Figure 3.6. B) shows abduction subtracted from forward flexion.



A

B

Figure 3.6 – A representation of the 2 x 2 Factorial model. (A) The first condition, upper left, all movement. (B) second condition, lower right forward flexion minus abduction.

As the regions of interest are within the gray matter, a gray matter mask was applied. Table 3.2 shows the cortical activations for forward flexion and abduction after Family Wise Error, p=0.05 is adopted to correct for multiple comparisons along with a minimum voxel activation of 10 voxels. The table shows the high level of statistical confidence through both the clusters levels and peak activation coordinates. Table 3.3 shows the regions of interest and level of activation in a simpler format.

Figure 3.7 shows the activation patterns, shown in Table 3.2, at a second level, demonstrating the motor cortex and other activations

within the section. When the contrast was run to define the difference between forward flexion and abduction, at a Family Wise Error p=0.05, with no minimum number of voxel activations, no activation remained.

Table 3.2. Table showing the voxel activation of all movement as part of the pilot project. The Cluster and Peak levels are shown both with FWE and FDR.

Cluster p(FWEcorr)	Number of Voxels	Peak p(FWEcorr)	Peak T	X	Y	Z	Brodmann Area
0	4679	0	16.32	-4	-8	64	5
0	743	0	11.7	8	-6	68	5
0	713	0	9.73	-18	-18	18	
0	739	0	9.05	50	-32	20	13
0	109	0	8.21	22	-18	68	5
0	84	0	7.78	-54	-60	10	39
0	156	0	7.77	58	10	-6	22
0.001	39	0	7.4	34	-2	56	5
0.001	43	0	7	-6	58	-6	10
0	69	0	6.94	60	12	16	44
0	58	0	6.91	38	-70	-20	19
0.001	39	0.001	6.47	16	-12	16	
0.002	27	0.001	6.4	54	6	38	9
0.002	28	0.001	6.31	40	-52	-24	37
0	54	0.001	6.31	-22	-84	38	19
0.009	11	0.001	6.31	14	-36	54	5
0.001	33	0.002	6.22	-16	14	-10	Putamen

0.003	23	0.002	6.2	52	-66	6	37
0.004	20	0.002	6.13	-46	40	14	46
0.005	16	0.002	6.1	-32	58	-6	10
0.004	18	0.006	5.84	4	56	-10	11
0.008	12	0.008	5.73	-6	18	-8	25
0.004	18	0.01	5.66	-30	8	50	6
0.007	13	0.01	5.65	32	-50	60	7

Table 3.3. Table showing the regions of interest and the level of activation for both forward flexion and abduction

Region of Brain	Brodman Area	Number of Clusters	Number of Voxels
Frontal	5	5	5581
Frontal	6	1	17
Pareital	7	1	13
Frontal	9	1	54
Frontal	10	2	59
Frontal	11	1	18
Frontal	13	1	739
Occipital	19	1	58
Temporal	22	1	156
Limbic	25	1	12
Temporal	37	2	51
Temporal	39	1	84
Frontal	44	1	68
Frontal	46	1	20

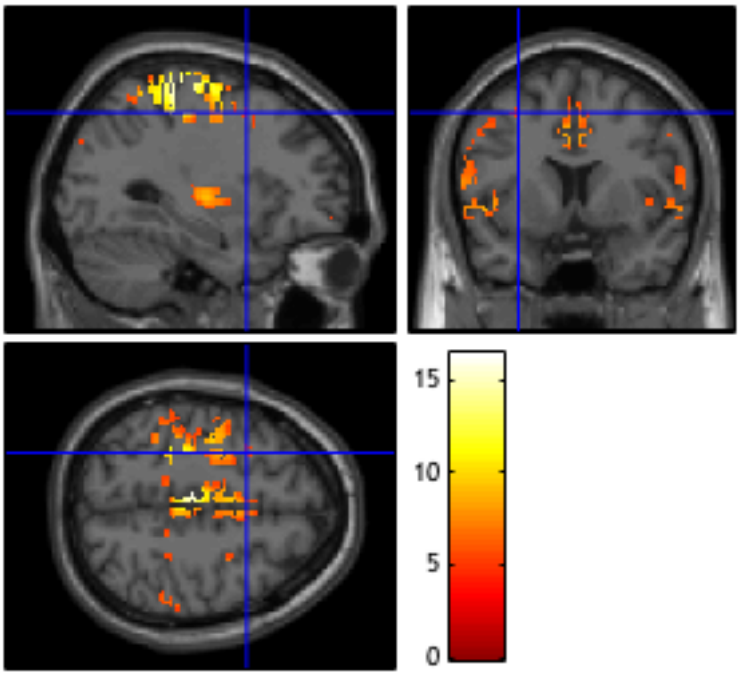


Figure 3.7 Graphical representation of the cortical activation in participants engaged in forward flexion and abduction. The blue cross shows the location of the motor cortex.

3.1.5.2 Motion Capture Study

Figure 3.9 shows the range of motion that was possible within the 1.5 T scanner. The first graph (Figure 3.9. A) Illustrates a range of motions of approximately 30° for forward flexion and extension. The second graph (Figure 3.9. B) demonstrates a range of motions of approximately 15°.

Figure 3.10 illustrates the means and the standard deviations of abduction/adduction whilst a participant was undertaking a cycle of 10 movements as described in 3.1.4.1.

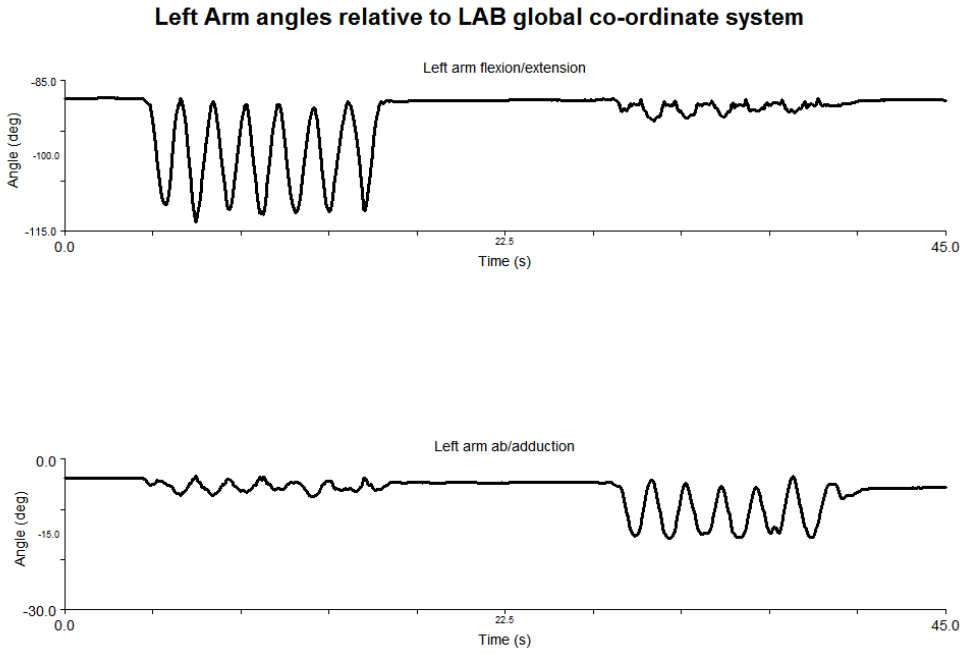


Figure 3.9. Graph showing the extent of upper limb movement possible within the 1.5 T Siemens scanner, A shows the movement flexion/extension, B shows the movement of abduction/adduction

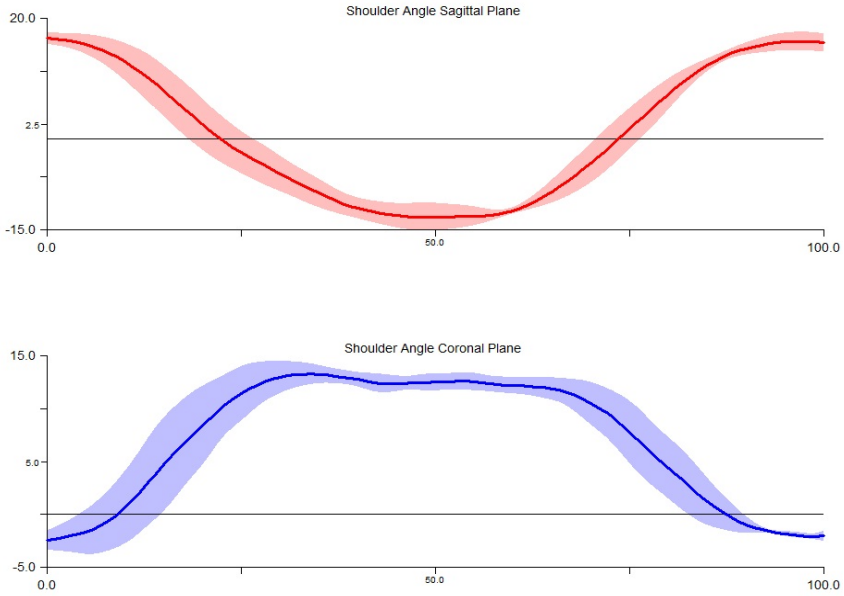


Figure 3.10. Graph showing the mean (thick dark blue line) and the standard deviations (light shaded area) in abduction/adduction whilst the participant was in the1.5 Tesler Siemens scanner

3.1.6 Discussion

3.1.6.1 fMRI Study

The movement paradigm (Figure 3.1) produced the expected activations, consistent with the areas set out in the literature review, Chapter 2.2. The main activation was in Brodmann area 5, involved in somatosensory processing and consistent with other upper limb movement studies [264]. Other Brodmann areas involved in movement were activated, area 6 premotor cortex; area 7 somatosensory association cortex; area 9 and 46 dorsolateral prefrontal cortex [265]. Brodmann area 44 is thought to be in be

involved in tasks that need a decision whether to proceed or not, consistent with the design paradigm [266].

Other expected areas of cortical activation were seen, including area 19 visual cortex responsible for processing visual information [267]. Although the participants used ear plugs, there was activation of the superior temporal gyrus, area 22, which is responsible for processing sound [268].

Brodmann area 25 was activated; this can be activated in both decision-making or when an individual is endeavoring to control fear [269]. Although an MRI scan is a routine medical investigation and widely used research tool, it can provoke anxiety in some subjects.

3.1.6.2 Motion Capture Study

The data (Figure 3.9) illustrates a range of motion of approximately 30° for forward flexion and extension was achievable. Further, a range of motion of approximately 15° was possible in abduction and adduction.

There was a certain degree of variation in movement in achieving in the movement tasks (Figure 3.10).

3.1.6.3 Implications for study

The results confirmed this as a valid paradigm to study movement in the scanner with a reasonable range of movement. A robust statistical test showed that it was not possible to distinguish between forward flexion and abduction, however, given the small numbers involved in the study it was thought appropriate to continue to use both movements.

It was noted that despite participants being given instructions as to the movements that should be undertaken, compliance remained an issue. Thus for the main study it was resolved that great pains would be taken to explain to the participants what was expected, and show them the passive movement whilst on the scanner table prior to the commencement scanning. During the scan if there was a failure to comply with the standard movement instructions then the scan was stopped and the participant shown a second time.

Some evidence of anxiety was noted in some of the participants, thus timing slots for the main scanning sessions were adjusted to allow sufficient time for explanation and familiarisation.

The results showed that the paradigm itself produced activations that needed to be factored into the analysis of the main project data.

The range of motion demonstrated by the use of the motion capture system showed a restricted range of motion, which would be important in drawing conclusions from the main study

3.2 EMG Method Development Study

3.2.1 Introduction

The EMG techniques are set out in 1.4.3. However, it was important to understand if these were effective at measuring shoulder movement and whether there were any technical issues. Following the study the nature of the movement was changed in light of the results.

3.2.2 Objective

The objective of the EMG method development study:

1. To ensure that thus setup allowed collection of technically adequate data.
2. To assess the practicalities of the proposed shoulder movements whilst collecting EMG data.

3. To compared different signals from the same muscle in order to rationalise the number of surface electrodes.
4. To identify any technical issues with the equipment.
5. To ensure the data collection was achieved.

3.2.3 Participants

All the participants were university students or members of the university staff (Table 3.4), who had no history of upper extremity pathology. Prior to attendance at the data collection session, the participants had been provided with a Patient Information Sheet, Appendix 1. Before the session commenced the participants had the opportunity to ask questions and after which they signed a consent form if willing to proceed, Appendix 2.

Table 3.4. Table to show the demographics of the participants of the EMG method development study (n=9)

Age	25 (20-36)
Sex	4 Male / 5 Female
Side	9 Left / 0 Right

3.2.4 Method

Chapter 1 gives the background to the methods adopted for testing, signal processing and data processing. In common with all EMG studies there has to be rationalisation of which muscles and the part of those muscles that are studied. In the method development study a particular aim was to explore whether one or two surface electrodes where necessary to monitor the muscles, pectoralis major and latissimus dorsi (Figure 3.11).

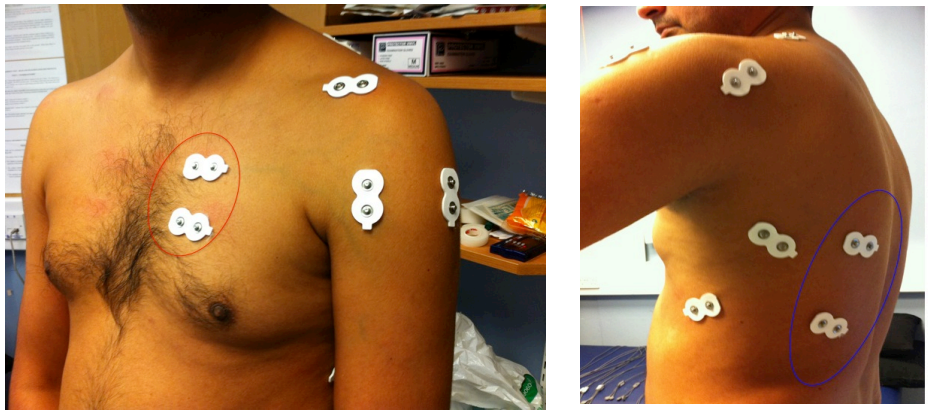


Figure 3.11. Photographs showing the placement of PM Sup and PM Inf (circled in red) and LD Sup and LD Inf (circled in blue)

The method development study has been limited to showing detailed activity of AD, MD, LD Sup and PM Sup in the thesis; this is to avoid the need of presenting excessive amounts of data. These 4 muscles have been selected, as they are of interest themselves, and are also representative of shoulder movement generally. The signals of AD, MD, PD, LD Sup, LD Inf, PM sup, PM inf, UT, SA, and

TM were recorded with surface electrodes and SSP, ISP and SUB by fine wire electrodes

The participants undertook four movements, set out below, at least 10 cycles were completed and throughout a metronome was used to standardise the frequency of the movement between subjects (1 Hz). The supine movements were undertaken in a constrained environment (Figure 3.12), with dimensions identical to those of the 1.5 T Siemens MRI Scanner, in which the fMRI work was to be undertaken. In all movements, phase 1 is the upward or lateral movement and phase 2 is the return movement.

Figure 3.12. Photograph showing the cardboard restrictor used to constrain the participants' arm movement whilst in the supine position.

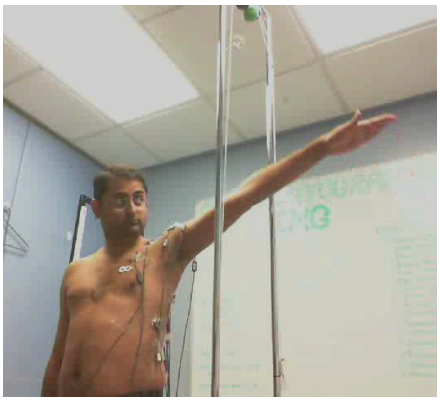


Figure 3.13. Photograph showing a participant undertaking the standing trial of abduction.

The four movements consisted of the following:

1. *Standing Forward Flexion*. Hand palm turned medially, starting vertically and through a range of motion of 180° and the returning to the original position.
2. *Standing Abduction*. Hand palm turned medially, starting vertically and through a range of motion of 180° and returning to the original position (Figure 3.13).
3. *Supine Forward Flexion*. Hand palm turned medially, starting horizontally with the couch and proceeding vertically through a range of motion of 30 degrees and returning to the original position.
4. *Supine Abduction*. Hand palm turned medially, starting horizontally with the couch and proceeding laterally in the horizontal plane through a range of motion of 15 degrees and returning to the original position.

3.2.5 Results

3.2.5.1 Standing Movement

3.2.5.1.a Forward Flexion

Table 3.5 presents the mean signal amplitude for the movement of forward flexion in phase 1 and the phase for all thirteen muscles during the 10 cycles. The standard error of measurement is presented along with the results of the paired test comparing both phases.

Figure 3.14 illustrates the activation pattern for AD, MD, LD sup and PM sup and Figure 3.15, shows the difference in activation patterns between PM sup, PM Inf, LD sup and LD inf.

Table 3.5. Table reporting the mean signal amplitude during phase 1, upward vertical and phase 2, downward movement of all thirteen muscles during standing forward flexion. SEM is the standard error of measurement. The t-test shown, assessed whether there was a difference between the two phases.

		Phase 1			Phase 2		
Muscles	n	Mean (%)	SEM	n	Mean (%)	SEM	t test *
AD	9	113.8	10.5	9	68.8	8.7	0.045
MD	9	116.4	13.1	9	63.5	12.8	0.073
PD	7	111.0	12.2	7	70.6	12.3	0.148
SUB	6	116.8	5.6	6	66.7	3.9	0.002
LD Sup	9	118.2	7.1	9	65.8	7.6	0.007
LD Inf	5	105.8	10.1	5	77.0	12.6	0.271
PM Sup	9	111.6	5.8	9	79.0	6.0	0.023
PM Inf	4	106.9	6.0	4	80.6	7.3	0.142
UT	6	111.8	13.5	6	73.9	14.3	0.227
SA	7	106.4	9.2	7	82.6	8.0	0.197
TM	8	108.0	8.9	8	75.6	9.0	0.111
SSP	3	93.4	14.9	3	116.4	32.6	0.652
ISP	5	104.7	17.0	5	83.2	15.3	0.538

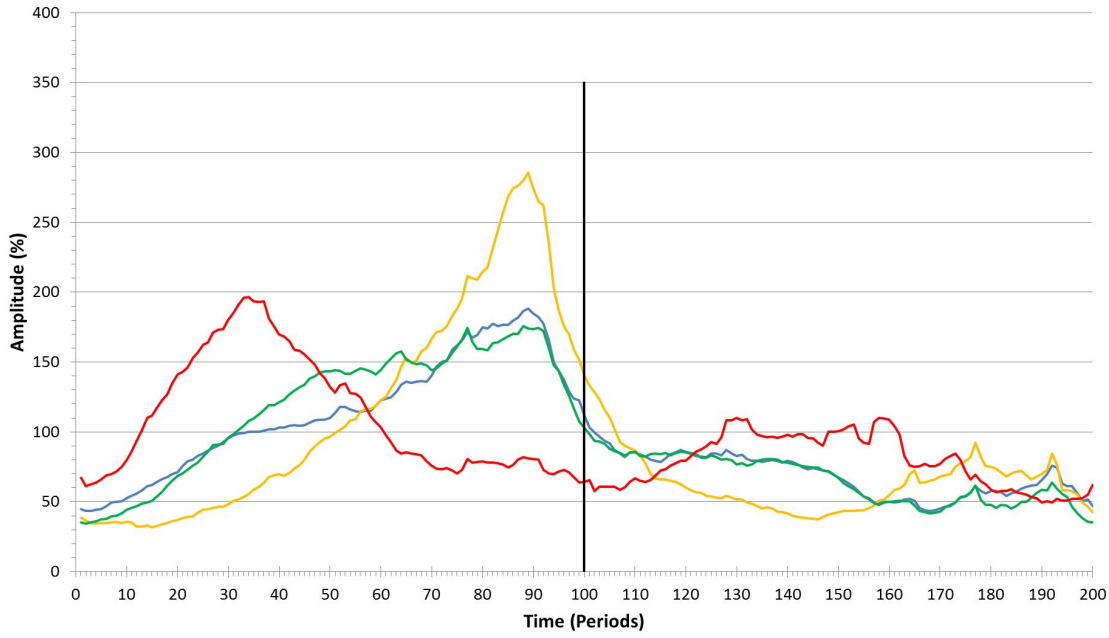


Figure 3.14. Graph to show the mean activation of AD (blue), MD (yellow), PM Sup (red) and LD Sup (green) during the time of the task, phase 1 (left) and phase 2 (right) during standing forward flexion.

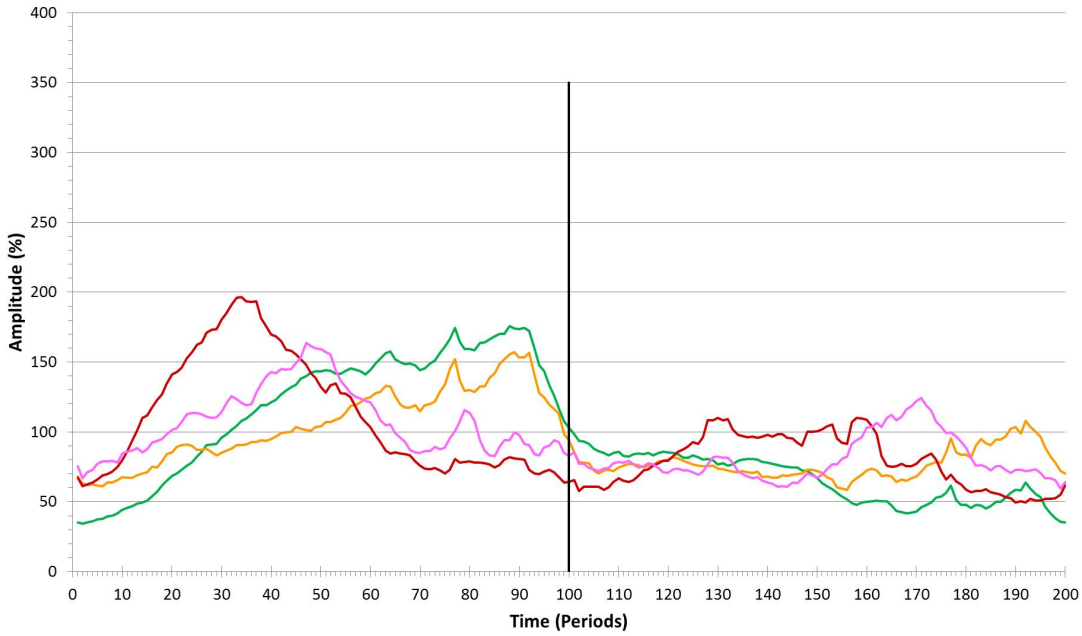


Figure 3.15. Graph to show the mean activation of PM Sup (red), PM Inf (pink), LD Sup (green) and LD Inf (orange/yellow) during the time of the task forward flexion whilst standing, phase 1 (left) up stroke and phase 2 (right) down stroke.

3.2.5.1.b Abduction

Table 3.6 presents the muscle activation for all thirteen muscles measured during abduction. It reports the standard error of measurement and the test value comparing the two phases. Figure 3.16 illustrates the muscle activations of AD, MD, PM sup and LD Sup. and Figure 3.17, shows the difference in activation patterns between PM sup, PM Inf, LD sup and LD inf

Table 3.6. Table reporting the mean signal amplitude during phase 1, upward vertical and phase 2, downward movement of all thirteen muscles during abduction. SEM is the standard error of the mean. The t-test assesses whether there was a significant difference between the two phases.

		Phase 1			Phase 2		
Muscles	n	Mean (%)	SEM	n	Mean (%)	SEM	t test *
AD	8	139.5	10.0	8	61.0	9.0	0.004
MD	9	140.5	10.5	9	58.8	9.6	0.003
PD	9	133.8	7.8	9	65.4	7.3	0.002
SUB Scap	8	122.0	9.0	8	65.4	7.6	0.004
LD inf	8	133.4	9.4	9	69.0	8.1	0.008
LD sup	4	123.5	6.8	4	71.9	3.2	0.012
PM inf	5	119.4	9.4	5	80.4	8.9	0.098
PM sup	2	112.7	6.4	3	80.9	5.0	0.264
UT	8	124.6	9.4	8	74.6	9.7	0.033
SA	8	119.4	5.7	8	78.3	6.0	0.008
TM	7	133.6	6.9	7	65.7	5.0	0.001
SSP	7	124.4	3.8	7	75.8	4.1	0.001
ISP	6	131.6	9.1	7	68.8	6.4	0.011

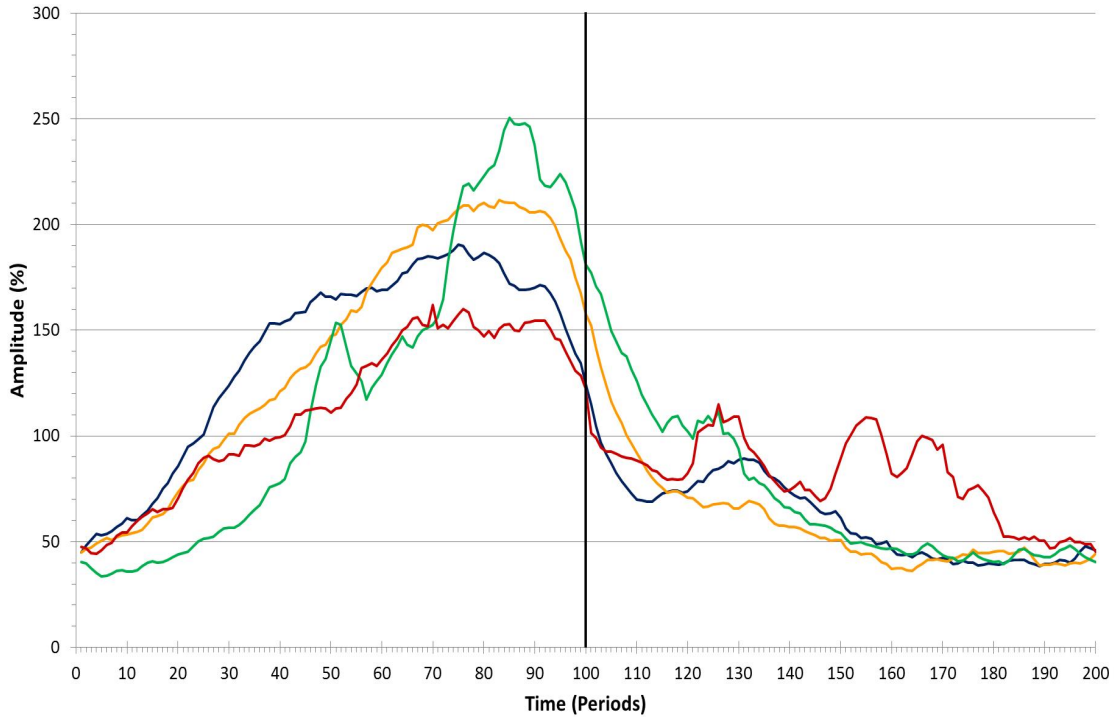


Figure 3.16. Graph to show the mean activation of AD (blue), MD (yellow), PM Sup (red) and LD Sup (green) during the time of the task, phase 1 (left) and phase 2 (right) during abduction.

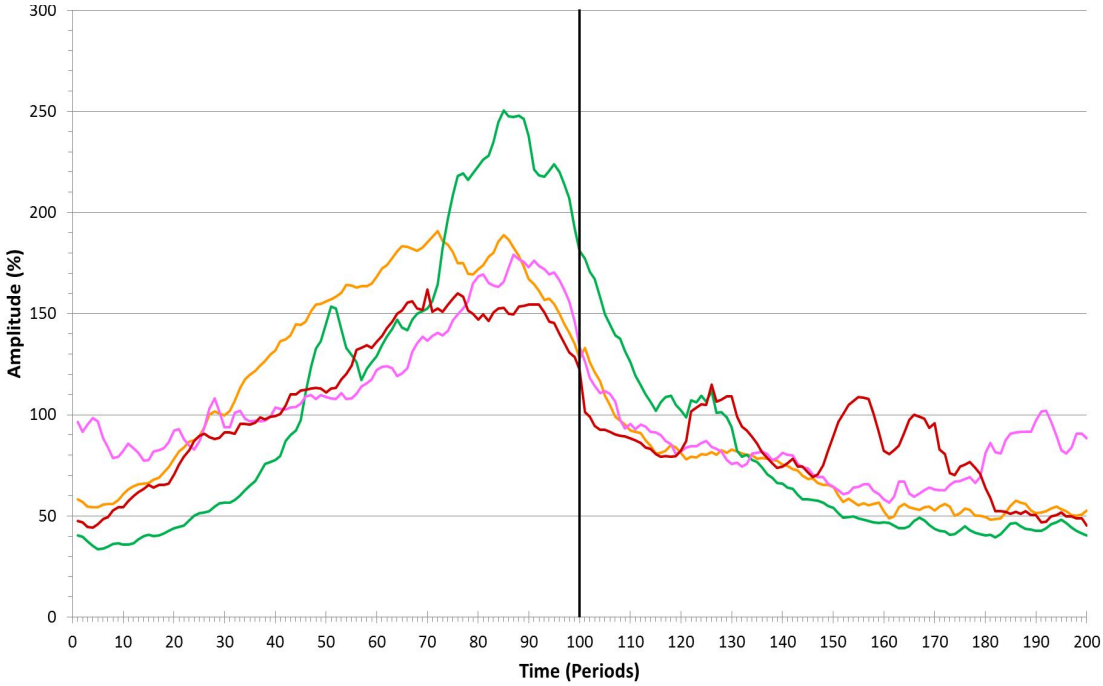


Figure 3.17. Graph to show the mean activation of PM Sup (red), PM Inf (pink), LD Sup (green) and LD Inf (orange) during the time of the task during abduction whilst standing, phase 1 (left) up stroke and phase 2 (right) down stroke during standing abduction.

3.2.5.2 Supine Movement

3.2.5.2.a Forward Flexion

Table 3.7, shows the muscle activations for all thirteen of the muscles tested whilst the participant was in the supine position. Figure 3.18 graphically shows the muscle activations for AD, MD, PM sup and PM inf.

Table 3.7. Table reporting the mean signal amplitude during phase 1, upward vertical and phase 2, downward movement of all thirteen muscles during supine forward flexion. SEM is the standard error of the mean. The t-test assessed whether there was a significant difference between the two phases.

		Phase 1			Phase 2		
Muscles	n	Mean (%)	SEM	n	Mean (%)	SEM	t test *
AD	8	129.5	5.1	8	66.6	4.3	0.000
MD	9	123.8	5.1	9	73.7	5.5	0.001
PD	9	111.9	6.4	9	85.0	6.5	0.068
SUB Scap	8	111.6	4.7	9	84.6	4.8	0.032
LD Sup	8	108.1	2.6	8	88.7	2.7	0.007
LD Inf	6	103.5	3.1	7	93.6	2.2	0.154
PM Sup	7	113.5	5.8	7	84.3	5.8	0.044
PM Inf	8	110.0	5.5	8	89.1	5.5	0.097
UT	3	113.0	3.2	3	83.2	3.6	0.049
SA	3	117.0	4.8	3	77.6	6.5	0.073
TM	7	109.4	3.9	7	90.4	3.6	0.043
SSP	9	108.3	3.4	9	88.4	4.0	0.026
ISP	7	116.3	5.0	8	80.8	5.5	0.012

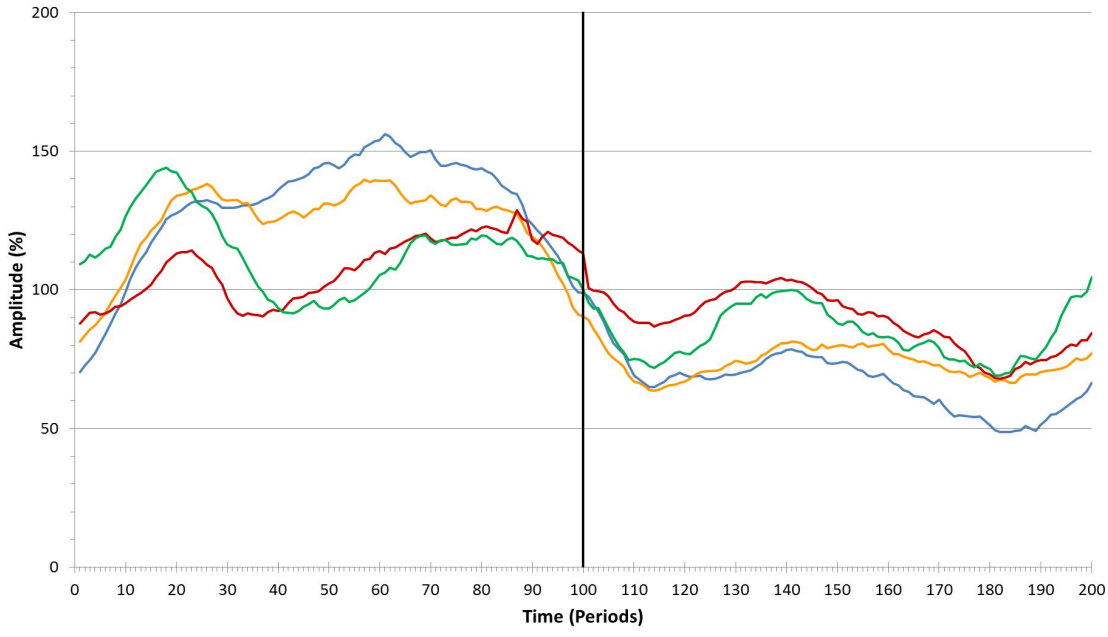


Figure 3.18. Graph to show the mean activation of AD (blue), MD (yellow), PM Sup (red) and LD Sup (green) during the time of the task, phase 1 (left) and phase 2 (right) during supine forward flexion.

3.2.5.2.b Abduction

Table 3.8 illustrations all thirteen muscle activations whilst the patient was in a supine position. Figure 3.19 shows the muscle activations of AD, MD, PM Sup and LD Sup.

Table 3.8. Table reporting the mean signal amplitude during phase 1, upward vertical and phase 2, downward movement of all thirteen muscles during supine abduction. SEM is the standard error of measurement. The t-test shown, assessed whether there was a difference between the two phases.

		Phase 1			Phase 2		
Muscles	n	Mean (%)	SEM	n	Mean (%)	SEM	t test *
AD	8	61.40	56.22	8	62.93	45.73	0.950
MD	10	41.28	22.25	10	40.23	22.60	0.932
PD	10	56.10	44.61	10	52.52	46.12	0.849
SUB Scap	8	44.21	53.08	8	41.84	48.82	0.708
LD sup	10	63.17	24.69	10	83.60	71.24	0.498
LD inf	10	84.95	46.01	10	71.69	33.06	0.472
PM sup	8	51.80	24.58	8	67.43	57.15	0.526
PM inf	6	68.40	18.65	6	50.52	29.29	0.170
UT	4	34.93	15.65	4	33.05	16.96	0.126
SA	2	35.45	10.39	2	24.65	4.88	0.500
TM	8	61.29	32.08	8	59.26	31.27	0.920
SSP	8	38.95	35.87	8	68.54	84.68	0.396
ISP	10	43.71	45.34	8	26.63	22.80	0.264

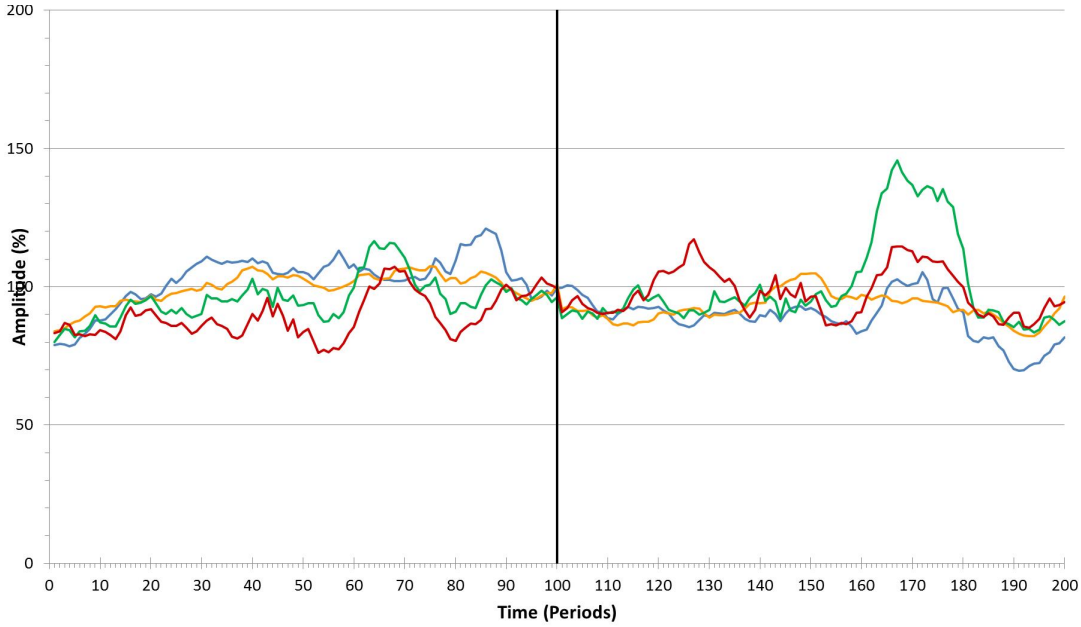


Figure 3.19. Graph to show the mean activation of AD (blue), MD (yellow), PM Sup (red) and LD Sup (green) during the time of the task, phase 1 (left) and phase 2 (right) during supine abduction.

3.2.6 Discussion

3.2.6.1 Standing Movements

As illustrated in Figures 3.14 and Figure 3.16 showed coherent patterns of muscle activation for both forward flexion and abduction.

In forward flexion, as expected and shown in both figures, AD and MD are primary movers of the shoulder joint [270]. The peak activation ratio of PM and MD was consistent with findings of Heuberer et al. [218].

3.2.6.2 Supine Movements

Figure 3.18 shows again coherent patterns of muscle activation for forward flexion that are consistent with the subjects being in a supine position. As discussed in Chapter 2.3 there is little work on muscle patterning whilst the individual is supine. However, applying first principles, the pattern shows activation of PM sup, AD and MD in phase 1, with stablisation of the humeral head by a coupling effect of LD sup. There is a second lesser peak in phase 2 of all four muscles, which would stabilise the humeral head and arrest the descent as the arms comes to rest on the couch.

The patterns of muscle activation for abduction in the supine position (Figure 3.19) is at first sight less distinctive and coherent. As can be appreciated the range of movement (Figure 3.9B) is considerable less and the nature of the movement in this position is consistent with the finding.

3.2.6.3 Implications for Main Study Protocol

The results were reviewed by a number of clinicians actively treating the intended patient group and it was felt that the majority of patients would be unable to achieve forward flexion or abduction

exceeding120°. This was taken into account in the final protocol, where forward flexion or abduction was attempted to 90°.

Muscle activation for abduction in the supine position (Figure 3.19) showed less distinctive patterns. However, these may be consistent with position and nature of movement, and so it was appropriate to measure this movement in the main study.

As discussed earlier, Chapter 1.4.3, where the muscles have different heads or varying fibers' orientation, the position for surface electrodes is complex. The surface electrode gives a measure of muscle activity at the sampled point, not necessarily representative of the muscle activity as a whole. In the muscles pectoralis major and latissimus dorsi, two fixed positions had been used, inferior and superior (Figure 3.11). Given the greater reliability of in the superior positioned electrodes, this was adopted in the main protocol.

During the supine measurements if was noted that a few participants experienced discomfort from the fine wire electrodes whilst getting into position. This was overcome by operator assistance and additional padding on the examination couch.

3.3 Reproducibility Study

3.3.1 Introduction

For any final results to be valid there needs to be confidence that the experimental design is reproducible. This is particular relevant in fMRI studies [271].

3.3.2 Method

One of the participants, aged 31, was invited to repeat the scan 13 months following the original scan. Exactly the same procedures were followed as if the subject was attended for the first time.

In the method development analysis, Brodmann areas 5, 6, 7, 9, 44 and 46 were identified as being activated whilst undertaking the paradigm. A cortical mask was then created based on these Brodmann areas and the first level analysis was computed. The contrast of both forward flexion and abduction was selected and a FWE (p0.05) with a minimum threshold of 10 voxels was selected.

3.3.3 Results

Table 3.9 shows a comparison of activation between the two scans, and Figure 3.20 shows the cortical activations of the second scan.

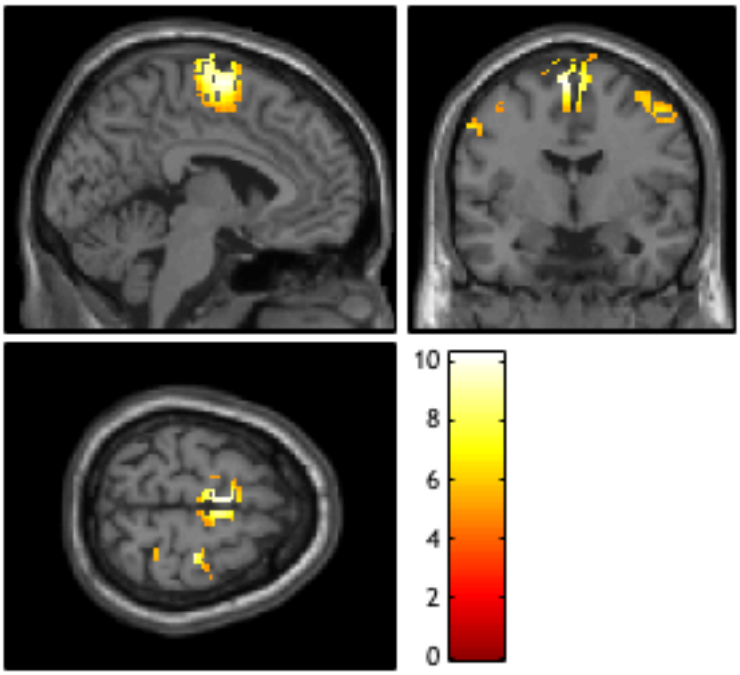


Figure 3.20. Graphical representation of the activation whilst undertaking both forward flexion and abduction

Table 3.9 - A comparison of activation of the same individual, with a gap of 1 year between fMRI analysis.

First Scan				Second Scan			
Brodmann Area	No. of Voxels	PFWER	T Value	Brodmann Area	No. of Voxels	PFWER	T Value
6	105	0.000	7.79	6	1356	0.000	9.67
5	40	0.000	9.60	6	603	0.000	10.21
6	26	0.001	5.77	6	64	0.000	6.96
				6	50	0.000	5.82
				6	32	0.000	5.71
				9	23	0.000	5.57
				7	13	0.000	6.02
				5	13	0.000	6.96
				5	11	0.000	6.06

3.3.4 Discussion

As shown (Table 3.9) there were comparable activations although the second scan produced far greater activations. However, activations were present for this individual in the appropriate Brodmann areas. Reassuringly the p valve remained at 0.001 or less after FWER was applied.

3.4 Main Study Methodology

3.4.1 Participants

The main study consists of two parts, the EMG study of normal shoulder movement (n=21) and the EMG/fMRI comparison study between patients with polar type II/III (n=16) compared to age matched controls (n=16). The former will be referred to as the 'normal shoulder study' and the latter as 'the comparison study'. The results and related discussions are set out in Chapters 4-6.

The normal shoulder study involved participants without any previous shoulder pathology and were recruited through the intranet at the University of Liverpool and consisted of both students and members of staff. All the participants were above 20 years of age.

For the comparison study, the patients were identified and recruited through the physiotherapy department at the Royal Liverpool and Broadgreen University Hospitals Trust (RLBUHT). The patients were examined within the specialist shoulder unit of the RLBUHT by the:

i. Senior professorial orthopaedic surgeon;
ii. Senior Physiotherapist, (with long history of treating should conditions).

Both of these individuals had to agree that the patients could be classified along the polar type II/III continuum before they were admitted onto the study. The patient group of the 16 patients represented the greatest clinical manifestations of polar type II/III. This type of selection was undertaken to increase the potential for identifying a common characteristic which the patient group, as previous no fMRI or larger scale EMG studies had been undertaken. As previously identified, the incidence of Polar type II/III is extremely low, and the Unit receives referrals from other shoulder units generally when patients present with pathology of more extreme in nature. The patient sample in this study represents virtually all the patients treated by the Unit over a 4-year period. Only one patient declined to be involved in the study due to a pregnancy and the development of an unrelated major condition. The following exclusion criteria were adopted:

i. Collegane Disorders, such as Ehlers-Danlos syndrome.
ii. Previous significant surgery.
iii. Previous Trauma.
iv. MRI exclusion factors i.e. pacemakers.
v. Neuromuscular conditions, Multiple Sclerosis.
vi. Any brain pathology that might affect the results.

The healthy control subjects were recruited again from the intranet at the University. All the participants were above 16 years of age, Table 3.10. None of the patients had been treated with psychoactive drugs which may have had a cofounding influence [272]. Prior to the commencement of the testing, all the patients and controls were asked about their medical history and drug history. None of the patients or the controls had any diagnosed psychological conditions.

St Helens & Knowsley Local Research Ethic Committee granted ethical approval for the above two research protocols. All the participants in both studies were provided with an invitation letter, a letter to their GP and a Patient Information Sheet. Prior to commencement of testing, the participants were given the opportunity to ask questions and were asked to sign a consent form.

3.4.1.1 Normal Shoulder Study

Twenty-one health subjects (11 women and 10 men) with no history of upper limb musculoskeletal problems volunteered to participate in the study (Table 3.10). The mean age for the normal shoulder group was 24 years (range 20-29), the mean body mass was 72.8 kg (range 50.4-105.3), and the mean height was 171 cm (range 154-184).

Table 3.10. Table to show the anthropometric parameters for the normal shoulder group

	Mean	Std. D	Range	Minimum	Maximum
Age	24	2.7	9	20	29
Weight (kg)	72.8	18.3	54.9	50.4	105.3
Height (cm)	171	8.9	30	154	184

3.4.1.2 EMG and fMRI Comparison Study

There were two groups for this part of the study, a control group (n=16) and a patient group (n=16), (Table 3.11). This was the number that forms the basis of the fMRI and EMG analysis. An additional two individuals had been recruited into the study, but had to be excluded due to incidental findings on their brain MRI scan that may have influenced the results.

The control groups of (16 women) were healthy volunteers who had no history of upper limb musculoskeletal problems. The mean age of this group was 23 years (range 16-31).

The patient group (14 women and 2 men) had been clinically diagnosed with shoulder instability type II to III. As set out in Chapter 2.1, this type of shoulder instability is a spectrum disorder, with less demarcation from definitive pathologies and often involves a psychological element. As part of the selection both the senior

surgeon and senior physiotherapist both had to agree that they well along polar type II/III Stanmore continuum. All the patients' were being treated as out-patients by the Shoulder Unit at RLBUHT. The stage of their treatment at the time of recruitment in the study varied, and thus the Oxford Shoulder Score and the Western Ontario Shoulder Instability Index were used as a validated pseudo-marker for the level of their disease; full details of these scores are set out in Chapter 3.4.4. As previously identified in section 2.1.1.5, their condition although capable of remission, tends to be relapsing and remitting.

Sample size is a critical issue in any study, as are the need to ensure the tests are valid and reproducible. There has to be a pragmatic approach to a study where there are a relatively small number of patients who truly fall within the group. Taking the work of Seighier et al.[273] and Thirion et al.[274] together it has been established that a sample size of 15 is unlikely to produce type 1 errors. This is a concern if further reduced, if known areas of the region of interest are used [273-277]. Desmond et al.[278] In their statistical power analyses found that that for liberal thresholds p=0.05, that 12 subjects were required in order to achieve 80% power at the single voxel level for typical activations. As formal power calculation could not be done, It was felt that recruitment of 16 patients was comparable to other fMRI studies [173, 194, 279, 280] and feasible with our patient population.

This was a patient group that presented a challenge to examine through fMRI. One concern was head movement, which has been identified by other examining patients with movement disorders [272]. Head movement can be estimated and used as a regressor when processing the data, Chapter 3.4.3.2b, but the starting point has to be developing strategies to prevent movement. Although within the head coil a subject's head is clamped (Figure 3.21), movement can still occur within the clamp. Thus one of the instructions given to the subjects was to refrain if possible from moving their head.

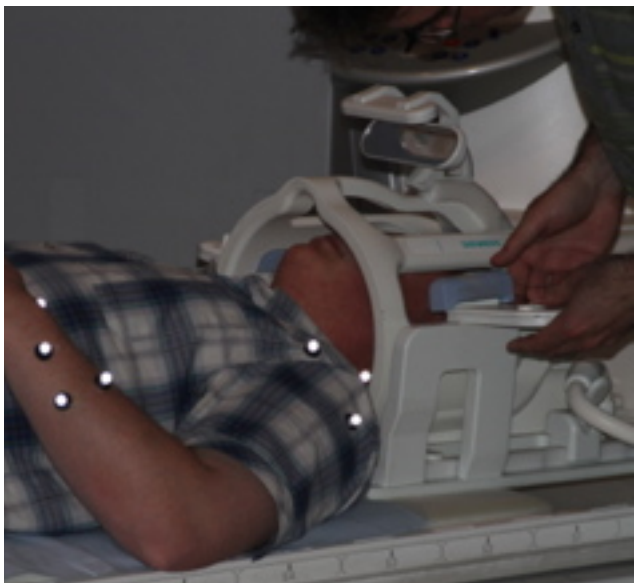


Figure 3.21. Photograph showing the side head clamp being positioned to reduce head movement within the head coil.

More information regarding the level of function and activity in the patient group is given in Chapter 4.1, where the results of the questionnaires are presented. The pain experienced by some of these individuals during activities involving the affected shoulder varied. However, from observation of some of the patients in clinic, it was apparent that most would not be able to achieve forward flexion or abduction greater than 90 degrees. Thus the normal shoulder protocol was modified to reduce these movements from 180 degrees to 90 degrees. It was considered better to increase the number of subjects who completed the task who had comparable data rather than increasing the range of motion. Anecdotally, it is suggested by some that instability may not exhibit itself until forward flexion or abduction is greater than 60 degrees. As the range of motion being tested is greater, at 90 degree, the method overcomes this potential criticism.

Table 3.11. Table showing the effected shoulder side for the patient group, the side tested in the control group, age range and average of the EMG/fMRI comparison study.

Patients	Age	Side	Controls	Age	Side
P1[2]	22	Right	AMC1	20	Left
P2	33	Left	AMC2	25	Right
P3	38	Right	AMC3	23	Right
P4	24	Right	AMC4	23	Right
P5	20	Right	AMC5	22	Right
P6	31	Right	AMC6	21	Right
P7	24	Left	AMC7	31	Right
P8	23	Right	AMC8	31	Right
P9	16	Right	AMC9	21	Left
P10	19	Right	AMC10	25	Right
P11	24	Right	AMC11	26	Right
P12	20	Right	AMC12	23	Right
P13	29	Left	AMC13	23	Right
P14	19	Right	AMC14	16	Right
P15	27	Right	AMC15	21	Left
P16	18	Right	AMC16	20	Left
Average	24.19		Average	23	

[2] P=patients, AMC=age matched controls

3.4.2 Study Design

3.4.2.1 Normal Shoulder Study

3.4.2.1.a Testing Protocol

The protocol was divided into two stages, the standing section and the supine section (Table 3.12). Both the forward flexion/extension and the abduction/adduction were undertaken to 180 degrees.

As in the method development study, the movement of the participant was restricted to the dimension of the Siemens 1.5 T scanner. A less invasive model was used, to assess patient compliance (Figure 3.22). A metronome was used to achieve consistent inter-subject movement frequency, being set to 1 Hz.

Table 3.12, Table showing the EMG protocol for the standing and supine testing

Standing		Supine	
Movement	Number of repetitions	Movement	Number of repetitions
Forward Flexion	15	Forward Flexion	15
Abduction	15	Abduction	15

Throughout all the EMG work described in this thesis, participants were closely observed. MacDermid et al.[281] developed stop criteria for individuals undertaking the FIT-HaNSA protocol. These were observed throughout the EMG testing and are as follows:

1. The participant stops or states it is too painful to continue.
2. The participant takes more than two beats to undertake the movement

3. The participant is generating the upper limb movement by using trunk/whole body for greater than 5 cycles.
4. The examiner believes the testing is placing the participant at risk of injury or suffering from an adverse complication if the protocol was continued.

Figure 3.22. Photograph showing a participant undertaking the forward flexion movement in the supine position.

3.4.2.1.b EMG Equipment

A TeleMyo 2400 G2 Telemetry System (Noraxon Inc., Arizona, USA), running MyoResearch XP software (Noraxon Inc., Arizona, USA),was used to acquire the signal and process of the data collected.

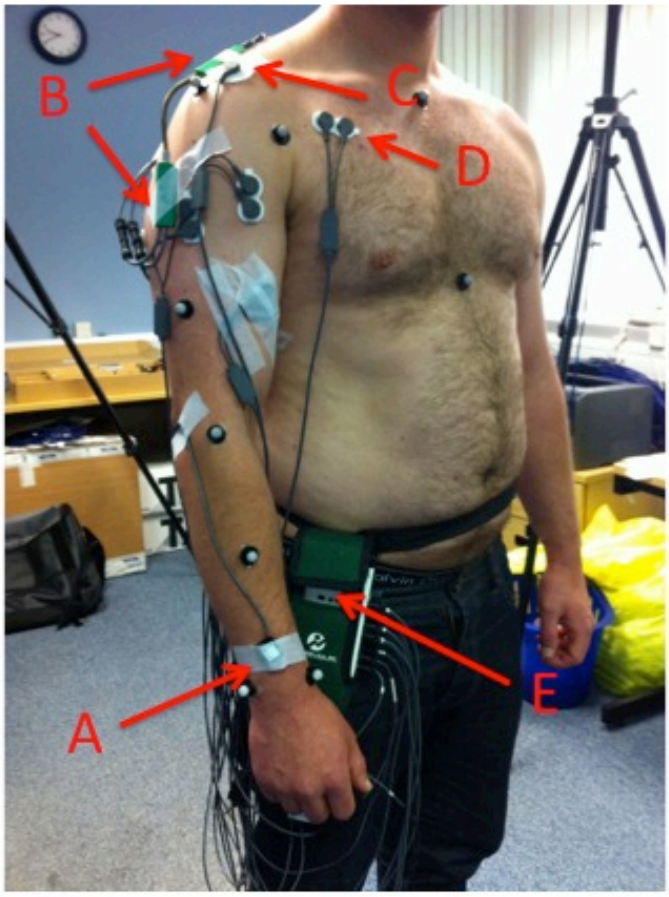
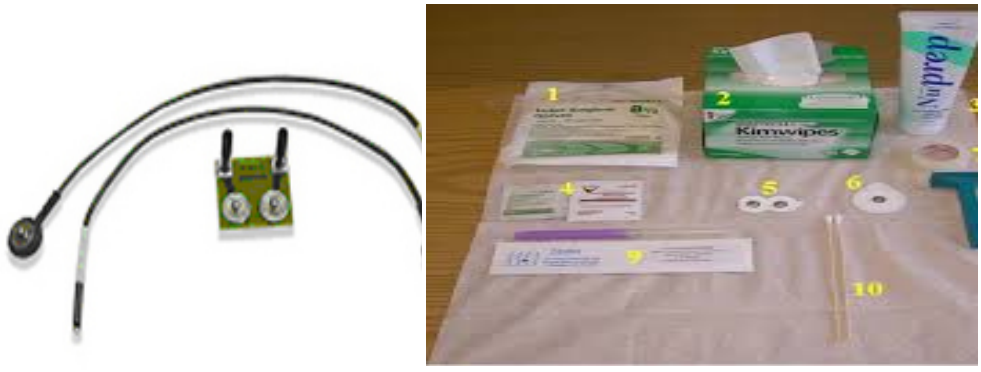


Figure 3.23. Photograph showing a participant from the normal shoulder movement study.
A – Accelerometer, B – Goniometer, C – Reference electrode, D – Surface electrode with EMG preamplifer lead, E – TeleMyo 2400 G2 Telemetry System within the waist pouch.

Two different types of bipolar electrodes were used, surface electrodes and fine wire electrodes [23]. Surface electrodes (Noraxon Inc., Arizona, USA) were self-adhesive pre-gelled silver/silver chloride, 8 shaped with an inter-electrode distance of 2 cm. Leads from the TeleMyo connected onto the surface electrode via snap-style connect (Figure 3.23, B) with a preamplifier proximal to the connection.

Fine wire electrodes were employed for the intramuscular recordings (Nicolet Biomedical, Division of VIASYS, Madison, USA). The fine wires were inserted into the muscle belly with a hypodermic needle, which is removed after wire insertion. The two 44 ga (0.05) insulated nickel alloy is insulated differentially at the end so as to measure local electrical activity over approximately 3 mm. The wires go into an EMG preamplifier, via springs mounted on the amplifier, which are then connected to the main unit via snap-on leads (Figure 3.24, A).



A

B

Figure 3.24. Both photographs showed equipment employed to undertake EMG data acquision. A shows a fine wire preamplification and leads. B shows individual items used in EMG data collection. 1 - Sterile Gloves, 2 - Tissues, 3 - Nuprep skin abrasive gel, 4 - Alcohol Wipes, 5 – Surface Electrode, 6 – Single Reference Electrode, 7 - Transpore surgical tape, 8 – Shaver, 9 – Fine Wire Electrode, 10 – Cotton Bud

Surface electrodes were used for the following muscles: AD, MD, PD, UT, SA, TM, LD, PM and BB. Prior to the application of the

surface electrodes the skin area was carefully prepared to reduce the impedance at the skin at the electrode site [282, 283]. Any hair was removed with a razor, the skin was cleaned with abrasive paste (Nuprep, weaver and Company, Aurar, Co, USA) and then the area was thoroughly dried [284, 285]. The muscles were manually identified and the surface electrodes were placed in line with the muscle fibers. The location and how the muscle was test is set out in Table 3.13, however the reference electrode was placed over the acromion (Figure 3.23, item C).

Fine wire electrodes were used for supraspinatus, infraspinatus and subscapularis (Figure 3.24, A, Figure 3.24, B item 9). The position of these fine wire electrodes was positioned in accordance with Table 3.14. Prior to needle insertion the skin area was cleaned with an alcoholic wipe. The needle was inserted aseptically. After the insertion the needle was removed, leaving the wires in situ. A number of isometric contractions were performed to embed the wires into the muscle belly [286-288]. The preamplifer was then secured to the skin with Transpore surgical tape (3M Healthcare Limited, Leicestershire, UK) (Figure 3.24, B, item 7) and the wire secured into the two springs on top of the amplifier. The wires from all the surfaces electrodes and the fine wire electrodes was then connected to the receiving unit, TeleMyo 2400 G2 Telemetry System (Figure 3.23, item E).

The EMG data was transmitted to the receiving Laptop wirelessly into the MyoResearch software, Version 1.8 (Noraxon Inc., Arizona, USA), which was subsequently used for data processing.

A 16G accelerometer (Noraxon Inc., Arizona, USA) and a 110 mm gonoimeter (Noraxon Inc., Arizona, USA) were also connected to the TeloMyo G2 Telemetry System. The accelerometer was positions on the dorsal surface of the wrist midway between the radial and ulna styloid processes (Figure 3.25, item A). The gonoimeter were fixed on the lateral aspect inferior and superior to the acromion (Figure 3.24, item B).

Figure 3.25. Photograph A shows 16G Accelerometer (Noraxon Inc., Arizona, USA). Photograph B shows the 110mm Goniometer (Noraxon Inc., Arizona, USA)

Before the start of the data acquisition, each muscle was manually tested and the output of the signal verified visually on the computer capturing the data.

The fine wire and surface electrodes signals were acquired simultaneously, and the sampling frequency rate and high frequency cut off were set to 1500Hz and the latter, 3000Hz [289]. The data processing guidelines of the International Society of Electrophysiology and Kinesiology were followed [290]. The band pass filtering was set to 10-500Hz for the surface electrode signals, and 10-1500Hz for the fine wire signals when the data was processed after completion of data acquisition. The MyoResearch software undertook a simultaneous video recording. The goniometer and accelerometer data was critical improving the accuracy of identifying the different phases of the movement cycle. This was particularly helpful when EMG data was being analysed for the supine position where the data identified the change in direction.

Table 3.13. Table to show the placement of surface electrodes with the movement that was undertaken in order to assess accurate placement[22, 291].

Muscle	Type of Electrode	Landmark	Manual Muscle to Verify Placement
AD	Surface	The clavicle was palpated. The electrode was placed on the anterior aspect of the arm 4 cm below the clavicle in line with the muscle fibers.	With the elbow a 0 degrees parallel to the trunk the patient was asked to forward flex against resistance.
MD	Surface	The acromion was palpated. The electrode was placed 3 cm below this landmark in line with the muscle fibers.	With the elbow a 0 degrees parallel to the trunk the patient was asked to abduct against resistance.
PD	Surface	The spine of the scapula was palpated. The electrode was placed 2 cm below the lateral border of the spin of the scapula in line with the muscle fibers.	With the elbow a 0 degrees parallel to the trunk the patient was asked to extend against resistance.

UT	Surface	The acromion and C7 were palpated. The electrode was placed mid-point between these electrodes in line with the muscle fibers.	The subject was asked to shrug their shoulders.
SA	Surface	The electrode was placed horizontally in the auxiliary area, anterior to the border of LD, at the level of the scapula	The patient was asked to forward flex their shoulder with the elbow at 0 degrees.
PM	Surface	The clavicle was palpated. The electrode was placed at an oblique angle just medial to the axillary fold.	With the shoulder and elbow at 90 degrees, against resistance the patient was asked to adduction their shoulder
LD	Surface	The inferior angle of the scapula was palpated and the electrode was placed 6 cm inferior to this point.	The subject was ask to abduct their arm and internally rotate. They were asked to extend against resistance.
	Surface	The electrode was placed	The patient was asked to

TM		over the muscle belly, immediately lateral to the lower one third of the lateral scapula.	adduct and internal rotate their arm against resistance.
BB	Surface	The patient was asked to gently flex their elbow to palpate for the muscle belly.	The patient was asked to flex their elbow against resistance.

Table 3.14. Table to show the placement of fine wire electrodes with the movement that was undertaken in order to assess accurate placement [22, 291].

Muscle	Type of Electrode	Landmark	Manual Muscle to Verify Placement
SSP	Fine Wire	The electrode inserted 1.5 cm above the mid point of the spine of the scapula. Inserted to depth of approximately 2.5 cm.	Against resistance with the humeral head internal rotation the subject was asked to abducted their arm in the scapular plane.
ISP	Fine Wire	The electrode was inserted 2.5 cm below the midpoint of the	With the elbow at 90 degrees flexion

		spine of the scapula. As with SSP, the needle was inserted to a depth of 2.5 cm. In the second part of the study this muscle was studied using surface electrodes. The spine of the scapula was palpated and the electrode placed 2.5 below the midpoint.	parallel to the trunk, against resistance the subject was asked to externally rotate their arm.
SUBS	Fine Wire	In order to lift the scapular off the posterior trunk, giving access to the muscle, the arm was placed behind the individuals back. The electrode was inserted 5 cm below the spine of the scapular in the direction of the underside of the scapular.	With the elbow flexed at 90 degrees and parallel with the trunk, against resistance the subject was ask to internally rotate their shoulder.

3.4.2.1.c Muscle Selection

It would be nice to study all the muscles involved in shoulder movement. However, even if this was technically possible, there

needs to be a more focus approach based on existing knowledge or reasoned suspicion based on clinical experience.

Within the Unit amongst the clinicians existed a large amount of experience of direct observation of the muscles that may implicate the shoulder instability of the Polar II/III patients. The experience is spread between 3 Consultant Orthopaedics Surgeons and 2 senior physiotherapists. The selection of the muscles was made on direct advice of those who have treated these patients' for a long period of time. Further, muscles which other units had advanced as causative of instability in these types of patients, where tested as they conflicted with our own experience, in particular PM and LD.

These muscles were used as a benchmark against which the fMRI and EMG patients' and controls would be compared. Thus the muscle selection was based on the muscles of interest particularly in the subsequent study.

3.4.2.1.c.i Pectoralis Major and Latissimus Dorsi

The clinical suspicion of a number of individuals involved in the study was that the instability was caused by defective activation in both pectoralis major and latissimus dorsi. In some patients this coupling effect of these two muscles has been found present. The dysfunction has thought to cause instability [215, 243, 292].

However, these two muscles are often omitted in studies and there is conflicting evidence as to their role [85, 232].

3.4.2.1.c.ii Supraspinatus, Infraspinatus and Subscapularis

The rotator cuff muscles are critical to maintaining shoulder stability, however, previous work has shown the unreliability of identifying the fourth cuff muscle, Teres Minor [293].

The role of both SSP and ISP in stabilising the shoulder becomes increasingly important as the angle of the upper limb increases, particularly in abduction [218].

The posterior cuff muscles such as SSP and ISP, along with TM are thought to have a synergistic action to prevent translational forces causing the shoulder to become unstable [234]. SUBs and SSP over activation has been recorded in shoulder instability patients [85, 244]. The rotator cuff muscles have also been shown to have a pivotal role in a compensatory stabilisation in pathologies such as rotator cuff tears [248].

3.4.2.1.c.iii Anterior Deltiod, Middle Deltiod and Posterior Deltiod

These muscles have a well-established role in both forward flexion and abduction. They have also been shown to have a correlation is one of the prime muscles of interest in our study, PM [218].

Further, inactivation of the PD has been shown in MDI patients as well as delay onset in action [244].

3.4.2.1.c.iv Serratus Anterior

This muscle has a role in stabilising the scapular, having greater activity in adduction compared to forward flexion [218]. McMahon et al. has suggested that this muscle is involved in shoulder instability [250].

3.4.2.1.c.v Teres Major

This muscle is active in abduction and has been shown to exhibit abnormal activation in response to other shoulder pathologies, such as rotator cuff tears [256, 294].

3.4.2.1.c.vi Upper Trapezium

This muscle has been shown to be activated in both forward flexion and thus inclusion was thought appropriate[234].

3.4.2.1.c.vii Biceps Brachii

This muscle has not been studied much in EMG studies looking at pathologies. However, inclusion was based on clinical observation in polar type II/III patients. The upper limb given the greatest number of degrees of freedom and number of joints have almost limitless paths of movement, these are particularly relevant when compensatory muscle action develops in response to pathology.

3.4.2.2 EMG and fMRI Comparison Study

3.4.2.2.a EMG Component

The same protocol was adopted as set out in Chapter 3.2, with the following differences:

i. The range of movement for the standing was reduced from 180 degrees to 90 degrees to enable more of the patient group to complete the exercise.

ii. Supine movements were not tested due to the cumulative effect of the movement within the scanner and the standing movement, which due to pain would not have been achievable.

iii. Fine wire electrodes were not used due to the increased discomfort of insertion may have led to non-participant or further abnormal movement following the experience of pain. Thus the muscles that were tested were AD, MD, PD, LD, PM, UT, SA, TM

3.4.2.2.b fMRI Component

The design of this study was a hybrid between a block design and an event related design. The design of the method development study was adopted for the main study. Overall there were 20 blocks of movement, with the order of that movement being randomised into 10 different versions of the protocol. These were presented to the patient via Presentation (NeuroBehavioural Systems, California), with different colors projected onto the scanner indicating the three

conditions (Table 3.15). The scanner triggered the Presentation software automatically, through the TPI signal. Each block was 12 seconds, comparable with other studies of a mixed block/event-related design [295-297]. Although a longer block time may have increased the sensitivity, it may also have introduced confounding temporal signal drift [272]. Regard had to be paid to the type of patients, as even these relatively small movements induced pain and risked shoulder dislocation.

Table 3.15. Table indicating the colors that were projected onto the scanner to indicate the movement required.

Red	Forward Flexion
Blue	Abduction
Green	Rest

As with the method development study, the patients were requested to undertake the movement at 1 Hz, and passively shown what this represented before being slid back into the scanner.

The functional MRI images were acquired on the Siemens1.5 T scanner, with an 8-channeled head coil. Some of the subjects had their structural T1 and T2 scan undertaken on the Siemens 3 T scanner, to enable multiple subjects to be examined on the same day. The structural scans were obtained not only for the data processing, but for ethical/governance reasons to ensure the subject did not possess a brain lesion. Although this may affect the fMRI result, more importantly these patients would require further investigation. In accordance with unit policy and the terms of the ethics approval, T2-weighted images were reviewed by a neuro-radiologist at the Walton Centre for Neurology and Neurosurgery. Functional scans were obtained with the scanner setting set out in Table 3.16.

Table 3.16. Table to show the scanner settings during the functional MRI data acquisition.

35 Slices
Field : 192 mm x 192 mm 100 FoV Phase
Slice : 3 mm 0.6 mm gap
TR 3000
TE 45
Transversal Orientation
Acquired AC/PC
Flip Angle 90 degrees
64 x 64 resolution
Order of acquisition : Ascending
164 measured
Echo Space 0.62 ms
EPI Factor 64

3.4.3 Data Processing

3.4.3.1 Normal Shoulder Study

3.4.3.2 EMG and fMRI Comparison Study

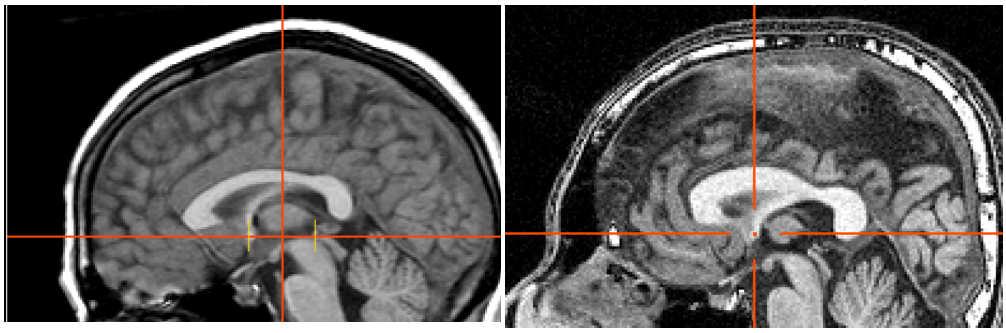
The data was processed with SPM in accordance with the principles set out in Chapter 1.4.2. The following is a detailed method as adopted to the data set out.

The EMG data was processed in the same manner as in the normal shoulder study, Chapter 3.2.

3.4.3.2.a Pre Processing

The Dicom files were imported into SPM. As some of the subjects had their structural scans undertaken on the Siemens 3 T scanner the anterior commissure was manually entered on the functional MRI images and the structural, T2 images. The scanner software assumes the anterior commissure to be at the centre of the skull, whereas it generally is more anterior and inferior (Figure 3.26). The

anterior commissure is used by SPM as a landmark during processing.



A B

Figure 3.26. Two sagittal slices. A shows the centre of the skull and the point marked as the anterior commussure by the scanner software. B shows the true anterior commussure.

The patient's affected side was used for the movement protocol, which resulted in a small number of left-sided subjects. In order to increase the number in the patient group, prior to preprocessing the images of left sided subjects were flipped. A symmetrical version of the template was created. Achieved by creating an averaging of the flipped and un-flipped version, which was used during the 'normalised' and 'segment' modules (Figure 3.27).

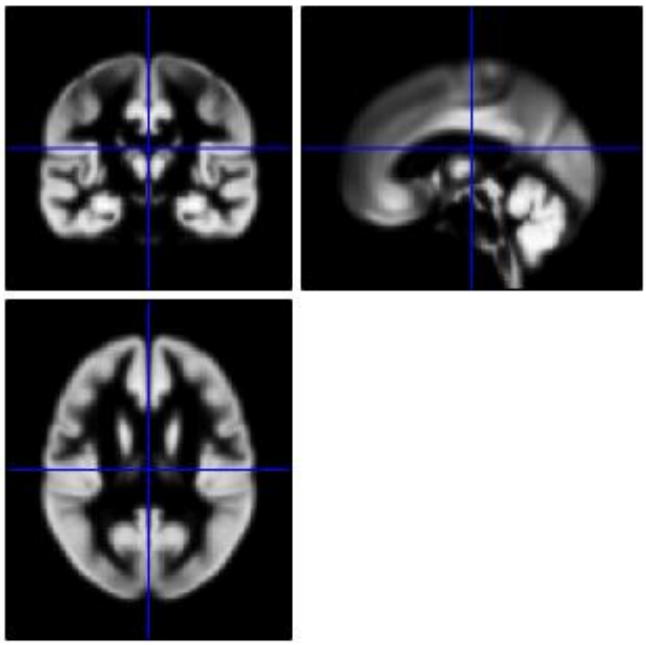


Figure 3.27. Sagital, coronial and transverse view of the symmetrical template that was created for the pre-processing steps of the data analysis.

3.4.3.2.b First Level Modeling

The principles behind this modeling are set out in section 1.4.2.2, but here I set out more detail of the specific steps taken to analyse the data set shown and discussed in Chapter 1.4.2.

There were ten different random orders created for the sequence of movement, these different time onsets were set up within SPM12. A High-pass filter of 128 was used; slice-timing correction to the first slice was performed within SPM12's Fourier phase shift interpolation. The six movement parameters created during the pre-processing stage were used as multiple regressors, these can be

observed in the six columns to the right of the 3 conditions (Figure 3.28).

At the first level the following contrast were created; all, rest was subtracted from forward flexion and abduction (1 1 -2); ff v ab, abduction was subtracted from forward flexion (Figure 3.29)

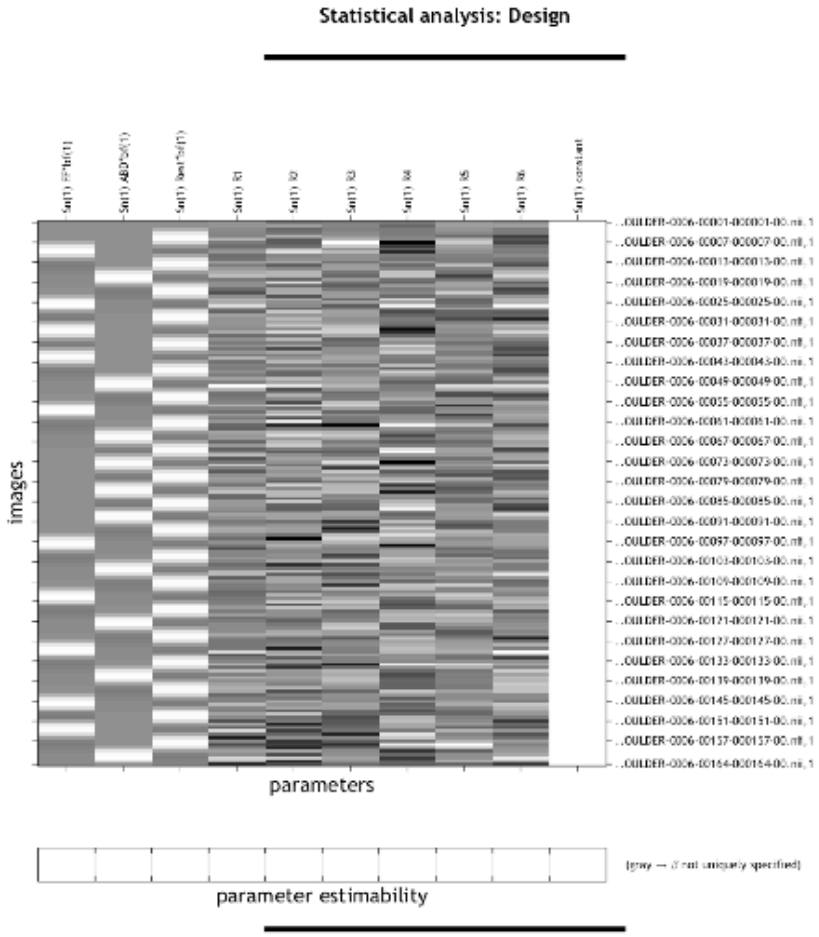


Figure 3.28. Graphical representation of the 1st level model. The first three columns show the three conditions, forward flexion, abduction and rest. The subsequent column shows the six regressors.

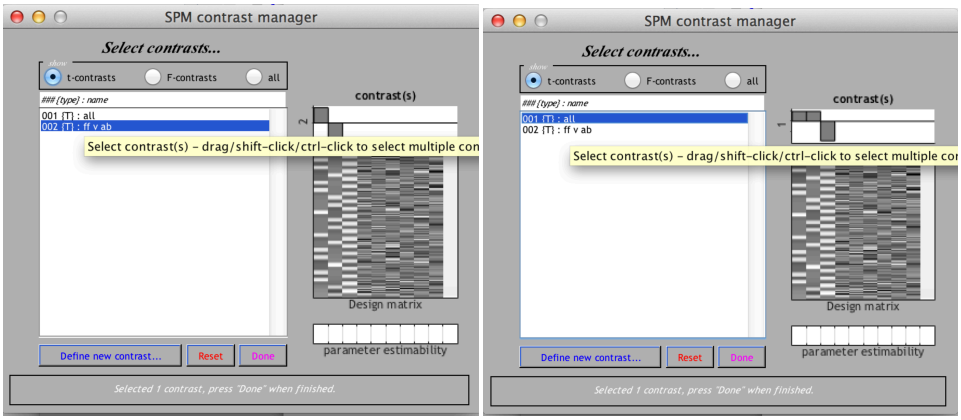


Figure 3.29. Screen print from the contrast manager of SPM. The testing contrasts all movement and subtracting abduction from forward flexion.

3.4.3.2.c Second Level modeling

In the main study the second level modeling two types of analysis were undertaken. The two factorial models were created to enable comparison of the two contrasts, all movement versus rest and abduction subtracted from forward flexion. The same type of modeling was followed as in the method development study (Figure 3.3). Masks were used to reduce the search space. The two masks that were used in the method development study were utilised, namely, the grey matter mask and regions of interests based on the results of the pilot method development.

In addition, further modeling was undertaken using the two scoring systems, the Oxford Shoulder Score and Western Ontario Shoulder Instability Index, Appendix 3 and 4.

As with the method development study Family Wise Error rate was used to control the chance of one or more false positives within all the detected voxels [272].

3.4.4 Questionnaires

All the participants in the fMRI and EMG comparison study completed three questionnaires (Appendix 3, 4 and 5). The questionnaires were completed before the commencement of the testing.

The patient group is extremely difficult to classify at their respective points between polar type II and III. The questionnaires of the Oxford Instability Score and the Western Ontario Shoulder Instability Index, where chosen to address the participants' functional status. The Beck's Depression Inventory was used to assess psychological parameters of the participants.

3.4.4.1 The Western Ontario Shoulder Instability Index

The Western Ontario Shoulder Instability Index was first published by Kerkley et al in 1998 [298]. It was designed to be used as an outcome measure of patients with shoulder instability recruited into

clinical trials [299]. It was developed and evaluated employing methodology developed by Kirschner and Guyatt, using a framework specifically aimed at assessing health indices [300]. The scoring system has been validated and is more responsive than other scoring systems such as the Constant Score [299]. This scoring system is regularly used for shoulder instability, most recently in Bateman et al. work [301].

3.4.4.2 Oxford Shoulder Instability Score

This was the second shoulder scoring system from Oxford. The original Oxford Shoulder Score was designed for shoulder operations other than stabilisation [302]. The subsequent scoring system, the Oxford Shoulder Instability Score, published in 1999[303], was designed to fill the gap left by the first questionnaire.

The basis of the questionnaire was interviews undertaken of 20 patients with shoulder instability. The scoring system has been validated and shown good comparative sensitivity to a patient's perception of their shoulder condition [299]. Like the Western Ontario Shoulder Score it performed better that the Constance Score in terms of reliability, responsiveness and validity.

3.4.4.3 The Beck's Depression Inventory

The Beck's Depression Inventory [304] is a widely used questionnaire across medical specialties [305]. It is not a diagnostic of clinical depression, as is the Diagnostic and Statistical Manual of Mental Disorders, but gives an estimation of the level of depression.

Recent work has shown a score of 13 or greater is 100% sensitive, with conflicting specificity of between 30-99%, with a positive predictive value of 0.72 [306, 307]. In the original work Beck advanced a score of 10 or greater as indicative of depression amongst medical patients [304]. It is a validated questionnaire in a number of settings including general medical patients and in general practice [308, 309].

It is suspected that there is a psychological component to polar type II/III shoulder instability, as originally proposed by Rowe [58] in the nineteen seventies. However, since then no real investigation has been undertaken to examine this aspect of shoulder instability and the Beck's Depression Inventory is considered a 'soft' diagnostic marker. However, there are no questionnaires for psychological illness screening in general; one has to have an idea of the condition you are suspecting first.

4 Functional MRI Results

Initially the results of the questionnaires will be presented to gain a comparative sense of the 16 patients and the 16 controls. Then fMRI data will be presented for each group and their activations compared. Conclusions to be drawn from the data will be reserved to Chapter 6.

4.1 Questionnaires

4.1.1 The Western Ontario Shoulder Instability Index

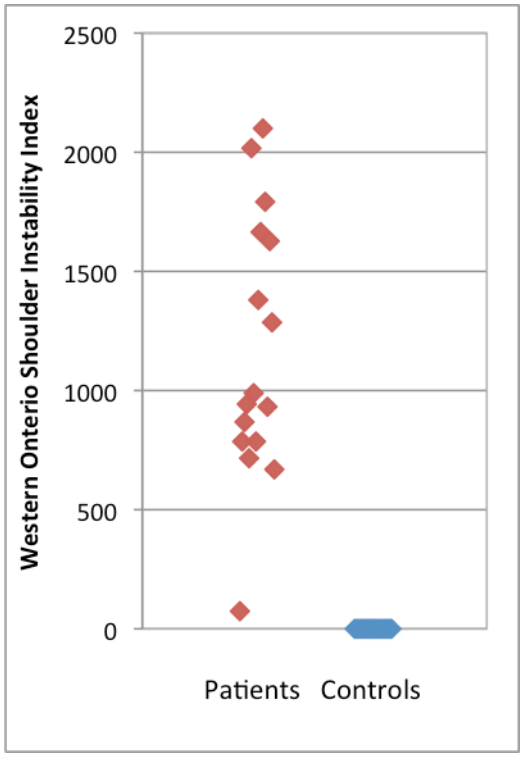


Figure 4.1. Graph to show the distribution of the Western Ontario Shoulder Instability Index for patients and controls, p=0.001

4.1.2 Oxford Shoulder Instability Score

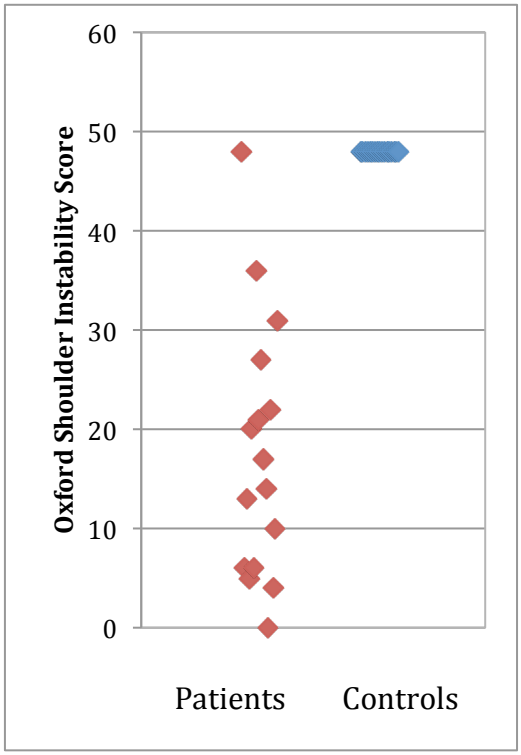


Figure 4.2 Graph to show the distribution of Oxford Instability Scores for patients and controls, p=0.001

4.1.3 The Beck's Depression Inventory

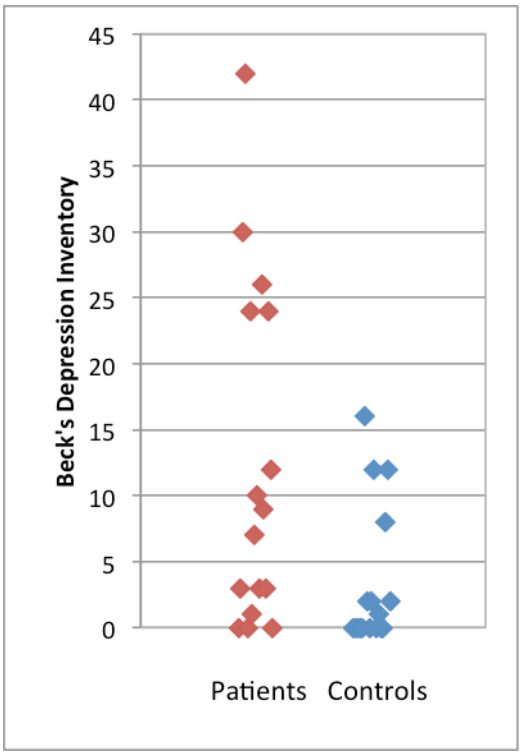


Figure 4.3 Graph to show the distribution of the Beck's Depression Inventory for patients and controls, p=0.001

4.2 fMRI Study

The issues surrounding which thresholds to be adopted in multiple comparisons has been addressed in Chapter 3.4.3, but all of the following results have been subject to a FWE of $p<0.05$.

Brodmann areas often contain a number of known functions and more likely some unknown. In reference to these areas mentioned, it would be a function that has been identified and that is specifically relevant to the study of movement.

There were 16 subjects in the control group and 16 subjects in patient group for the purposes of the analysis of the fMRI study. An additional two subjects had been recruited, however, one from the patient group and one from the control group as a result of the clinical MRI scans were discovered to have lesions that may influence their fMRI result. Adopting a conservative approach these individuals have been removed from both the fMRI and EMG analysis.

The Brodmann areas are taken from a combination of SPM Anatomy Toolbox (Version 2.1) [310-314] and WFU PickAltas [315, 316].

4.2.1 Global Results of Patients

4.2.1.1 All Movement

Figure 4.4 and Table 4.1 detail the results of all movements (both forward flexion and abduction) for the patient group, with an

inclusive grey matter mask applied. All 16 patients successfully completed the fMRI test.

Overall it can be seen there were 30 different activation clusters, with 11 clusters greater than 10 voxels with peak pFWE-corr <0.008, which are as follows:

i. Brodmann area 4, primary motor cortex, part of the precentral gyrus of the frontal lobe.
ii. Brodmann area 31, dorsal posterior cingulated cortex, upper part of the limbic lobe.
iii. Brodmann area 22, superior temporal gyrus located above the external ear.
iv. Brodmann area 44, pars opercularis, part of the inferior frontal gyrus of the frontal lobe, just anterior to the premotor cortex (Brodmann area 6).
v. Brodmann area 42, auditory cortex located in the anterior temporal gyrus of the temporal lobe, medial to Brodmann area 22.
vi. Brodmann area 3, primary somatosensory cortex, anterior to Brodmann area 2 which is anterior to Brodmann area 1, which is in turn adjacent to the central sulcus of the frontal lobe.
vii. Brodmann area 40, supramarginal gyrus, of the parietal lobe, part of Wernicke's area.

viii. Brodmann area 19, associative visual cortex located in the occipital lobe.

ix. Ventral lateral nucleus (VLN), located within the thalamus, which is situated between the cerebral cortex and the midbrain.

The voxel size is 2 mm^3 but inclusion of a single voxel is justified by the correction through multiple comparisons and through the robust Family Wise Error calculation, which implies that more levels of activation surround the voxel than survives the correction.

Confidence in the data in terms of type 1 errors can be grounded in the fact that these results survived Family Wise Correction, pFWE-corr <0.035 for the whole table, and pFWE-corr <0.01 if 10 voxels and greater are considered. The t-values range between 11.89 and 5.23.

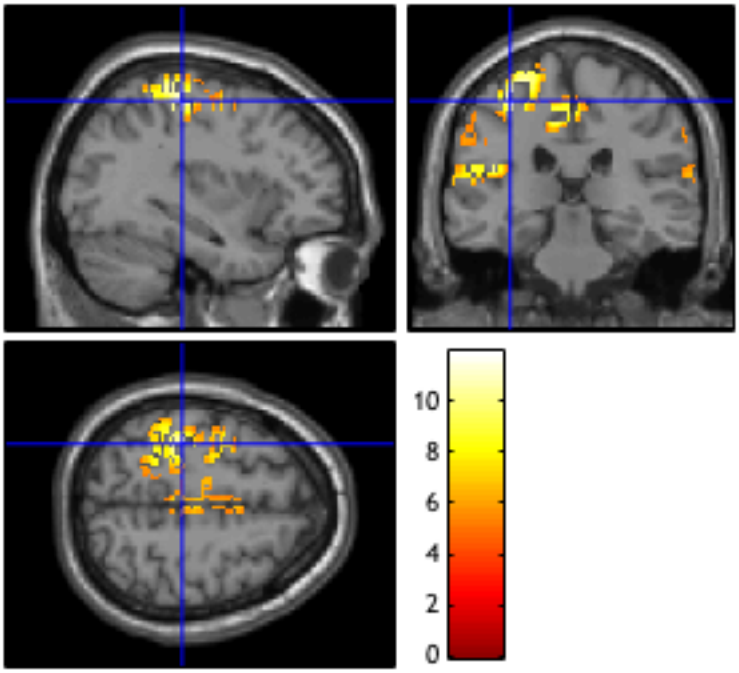


Figure 4.4 A graphical representation of the cortical activations for all movements. (Forward Flexion and Abduction) from the patient group, pFWE-corr 0.05.

Table 4.1 Table to show the cortical activations for all movements (Forward Flexion and Abduction) from the patient group, pFWE-corr 0.05.

			MNI mm			Brodmann's Area
Activated Voxels	p(FWE-corr)	T value	x	y	z	
3097	<0.001	11.89	-34	-28	56	4
325	<0.001	8.52	2	-28	50	31
131	<0.001	7.07	-50	12	-4	22
67	0.002	6.14	-60	6	12	44
52	0.001	6.49	66	-30	16	42
20	0.008	5.73	28	-36	66	3
19	0.002	6.2	-60	-42	26	40
13	0.002	6.21	-52	-64	12	19
13	0.002	6.13	-18	-18	18	VLN
10	0.002	6.09	62	-26	38	2

4.2.1.2 Forward Flexion

The activations for forward flexion in the patient group are reported in Table 4.2 and Figure 4.5. Prior to corrections, an implicit grey matter mask had been applied. There are 13 clusters of activation, less than the 30 (Table 4.1) for all movement. The level of activation above 10 voxels is as follows:

i. Brodmann area 4, primary motor cortex, part of the precentral gyrus of the frontal lobe.

ii. Brodmann area 31, dorsal posterior cingulated cortex, upper part of the limbic lobe.

iii. Brodmann area 13, insular cortex located in the posterior insular cortex within the frontal cortex.

iv. Brodmann area 6, premotor cortex and supplementary motor cortex, located anterior to the primary motor cortex (Brodmann Area 4) within the frontal cortex.

The levels of activations for abduction (Table 4.3) compared to forward flexion are higher (Table 4.2), with a number of voxels in abduction reaching the 1000 level mark compared to forward flexion at the 250 mark. It is abduction that is driving the higher levels of activation and numbers of clusters in the total movement (Table 4.1).

Confidence in the data in terms of type 1 errors can be found in the fact that these results survived Family Wise Correction, pFWE-corr <0.035 for the whole table, and pFWE-corr <0.01 if greater than 10 voxels are considered. The T valves range between 7.44 and 5.17.

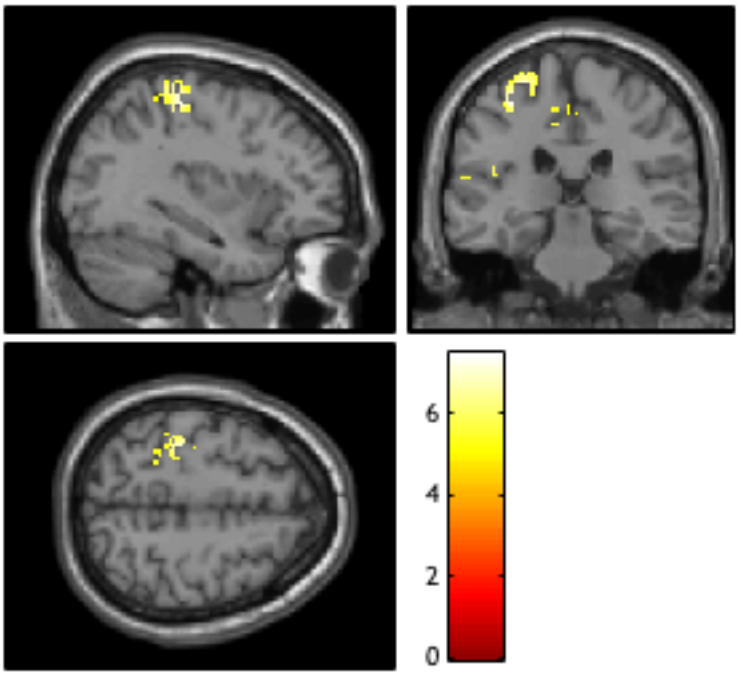


Figure 4.5 A graphical representation of the cortical activations for forward flexion from the patient group, pFWE-corr 0.05.

Table 4.2 Table to show the cortical activations for forward flexion from the patient group, pFWE-corr 0.05.

			MNI mm			Brodmann's Area
Activated Voxels	p(FWE-corr)	PeakT	x	y	z	
235	<0.001	7.44	-34	-28	56	4
35	<0.001	6.62	-6	-22	50	31
10	0.002	6.11	-44	-26	18	13
16	0.003	6.01	-4	-8	64	6

Figure 4.6 A graphical representation of the MNI co-ordinates, -60 -20 12 from the Anatomy Probability Atlas [1-5].

4.2.1.3 Abduction

Table 4.3 and Figure 4.7 show the activations for the movement abduction of the patient group. The analysis included a grey matter mask to reduce the number of comparisons. Overall it can be observed that there are 30 activation clusters, equal to the number for all movement (forward flexion and abduction) and double the number for the single movement of forward flexion. If the voxel threshold is raised to 10 then the number of clusters reduces to 8.

In the Brodmann areas that are greater than 10 voxels, at a peak and cluster level the pFWE-corr <0.004.

There are activations greater than 10 voxels in the following Brodmann areas:

i. Brodmann area 4, primary motor cortex, part of the precentral gyrus of the frontal lobe.
ii. Brodmann area 31, dorsal posterior cingulated cortex, upper part of the limbic lobe.
iii. Brodmann area 13, insular cortex located in the posterior insular cortex within the frontal cortex.
iv. Brodmann area 6, premotor cortex and supplementary motor cortex, located anterior to the primary motor cortex (Brodmann area 4) within the frontal cortex.
v. Brodmann area 43, part of the postcentral gyrus in the proximity to Brodmann areas 2, 6 and 40 [317].

Confidence in the data in terms of type 1 errors can be assured by the fact that these results survived Family Wise Correction, pFWE-corr <0.035 for the whole table and pFWE-corr <0.004, if analysis is limited to clusters with greater than 10 voxels. The t-values range between 9.89 and 5.15.

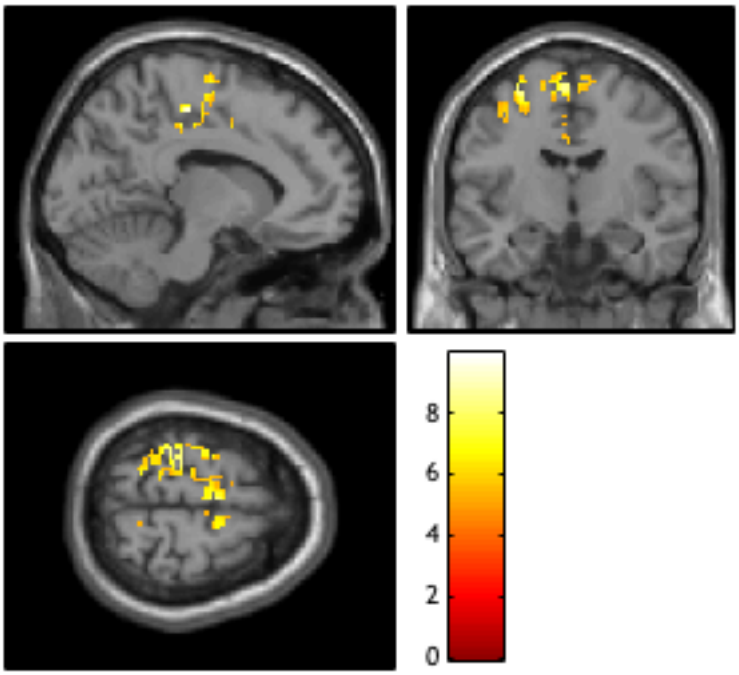


Figure 4.7 A graphical representation of the cortical activations for the movement of abduction from the patient group, pFWE-corr 0.05.

Table 4.3 Table to show the cortical activations for the movement of abduction from the patient group, pFWE-corr 0.05.

Cluster-Level		Peak		MNI mm			Brodmann's Area
p(FWE-corr)	Activated Voxels	p(FWE-corr)	PeakT	x	y	z	
<0.001	570	<0.001	9.89	-6	-22	50	31
<0.001	1018	<0.001	9.53	-34	-28	56	4
<0.001	277	<0.001	8.9	-44	-26	18	13
0.001	44	<0.001	7.13	2	-22	52	6
<0.001	58	<0.001	6.84	10	-6	68	6
<0.001	81	0.001	6.38	-64	-16	22	43
0.003	23	0.001	6.28	4	6	36	24
0	59	0.004	5.93	-50	12	-4	22

4.2.1.4 Abduction subtracted from Forward Flexion

When the contrast of abduction subtracted from forward flexion was modeled; an inclusive grey matter mask had been applied no voxels survived FWE correction. This finding is consistent with the findings in the method development study, section 3.1.5.1, which equally demonstrated no surviving activations within the same contrast. It

was thought that with the additional numbers of participants that due to the power increasing that some of the activations may survive.

Masks were constructed around the areas of interests demonstrated in the method development study, Brodmann areas 5, 6, 7, 9, 46 and 9. These where then used as inclusive masks to see if the reduced number of voxel comparisons increased the sensitivity. However, despite the increase in sensitivity, no voxels survived the Family Wise Correction.

The same contrast was modeled for the control group and no activation survived Family Wise Correction for multiple comparisons.

4.2.2 Global Results of Controls

4.2.2.1 All movement

Figure 4.9 and Table 4.4 detail the results of all movements of the control group (forward flexion and abduction), with an inclusive grey matter mask applied.

There were 36 different clusters of activation for the control group (n=16), this compares to the 30 different areas of activity in the patient group (n=16). If clusters of 10 voxels or greater are considered, then the patient group had (Table 4.1) 11 different clusters, whereas the control group had 17.

In summary, the control group had a greater number of clusters, but overall the patient group had an increase in the level of activation.

I will firstly consider the comparison of all movement without limitation to the number of voxels, and then consider the effects of limiting consideration to greater than 10 voxels. The control group (Table 4.4) had a mean level of voxel activation 90 (SD 267), with a range 1-1,545, and the cumulative total activation was 3,259. The

patient group (Table 4.1) had a mean level of activation 127 (SD 564), with a range 1-3096, and a cumulative total activation of 3,823.

If the consideration is limited to 10 voxels or greater, then for the control group (Table 4.4), the mean is 188 (SD 370) there is a range 10-1533, and the cumulative total activation 3,192. For the patient group (Table 4.1), the mean is 342 (SD 918) there is a range of 10-3,087, and the cumulative total activation is 3,763.

The following Brodmann areas can be distilled from the activation of the control group for all movement with a minimum level of activation of 10 voxels:

i. Brodmann area 6, premotor cortex and supplementary motor cortex.
ii. Brodmann area 13, insular cortex.
iii. Brodmann area 22, superior temporal gyrus.
iv. Brodmann area 3, primary somatosensory cortex.
v. Brodmann area 40, supramarginal gyrus.
vi. Brodmann area 19, associative visual cortex.
vii. Brodmann area 37, fusiform gyrus, located between the temporal lobe and the occipital lobe.
viii. Brodmann area 10, the anterior most part of the brain in the frontal cortex.

Confidence in the data in terms of type 1 errors can be grounded in the fact that these results survived Family Wise Correction, pFWE-corr <0.035 for the whole table, and pFWE-corr <0.008 if one restricts consideration to clusters greater than 10 voxels. The T values range between 13.41 and 5.21.

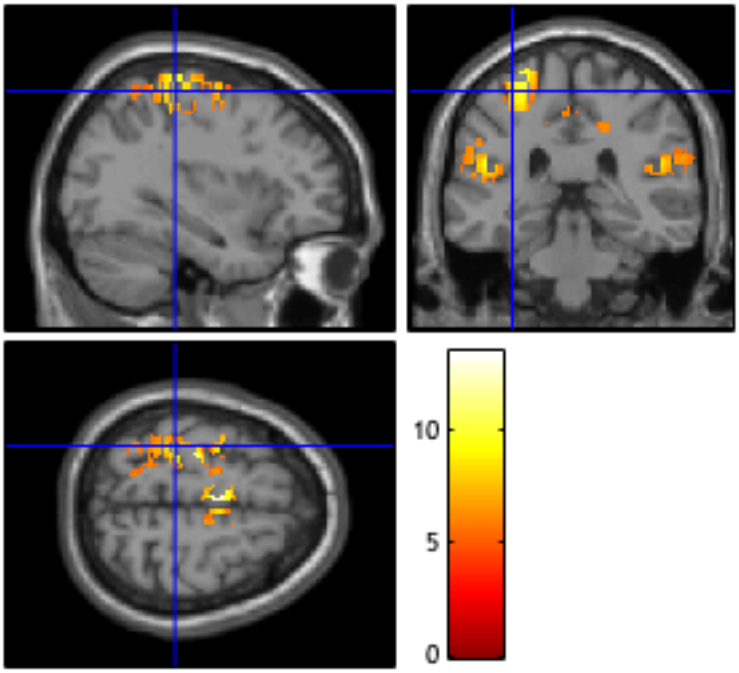


Figure 4.9 A graphical representation of the cortical activations for all movements of the (Forward Flexion and Abduction) in the control group, pFWE-corr 0.05.

Table 4.4 Table to show the cortical activations for all movements (Forward Flexion and Abduction) in the control group, pFWE-corr 0.05.

Cluster-Level		peak		MNI mm			Brodmann's Area
p(FWE-corr)	Activated Voxels	p(FWE-corr)	T	x	y	z	
<0.001	1546	<0.001	13.41	-4	-8	64	6
<0.001	345	<0.001	9.87	-44	-28	18	13
<0.001	184	<0.001	8.49	50	-32	20	13
<0.001	425	<0.001	8.36	-18	-22	12	13
<0.001	75	<0.001	8.13	16	-24	40	31
0.001	44	<0.001	7.61	22	-18	70	6
<0.001	97	<0.001	7.41	-50	12	-4	22
0.007	13	<0.001	6.76	-30	-24	50	3
0.001	38	<0.001	6.74	34	-2	56	6
0.006	15	<0.001	6.66	-54	-30	32	40
0.001	45	0.001	6.46	58	10	-6	22
0.001	31	0.001	6.41	56	2	36	6
0.001	32	0.001	6.32	24	-62	-16	19
0.001	38	0.002	6.13	-48	-68	0	37
0.003	22	0.005	5.88	52	-66	4	37
0.006	14	0.008	5.75	-6	58	-6	10

4.2.2.2 Forward Flexion

Table 4.5 and Figure 4.10 demonstrate the level of activation generated by forward flexion for the control group. Overall it can be seen that there are 21 clusters, compared to 13 in the patient group for the same movement (Table 4.2). If the analysis is restricted to clusters of more than 10 voxels, then the control group has 8 clusters compared to the patient groups' 6 clusters (Table 4.2).

Overall, in forward flexion the control group exhibits greater levels and numbers of cortical activation.

Considering the controls are in greater detail, if the size of the cluster is ignored, then the mean level of voxels is 38 (SD 71), the range is 251, and the overall level of activation is 811. If the analysis is restricted to clusters of 10 of more voxels, the mean increases to 97.5 (SD 89), the range is 234, and the overall level of activation is 780.

First considering all the clusters irrespective of size, the patient group (Table 4.2) had a mean level of activation of 30 voxels (SD 64), with a range of 234 voxels; overall the total level of activation is 393. If only the clusters are examined with 10 or more voxels, then the average increases to 73 (SD 98), with a range of 225 and the total level of activation is 365.

The table shows (Figure 4.5), pFWE-corr <0.035 for the whole table, and pFWE-corr <0.004 if one restricts consideration to clusters greater than 10 voxels. The t-values range between 9.51 and 5.19.

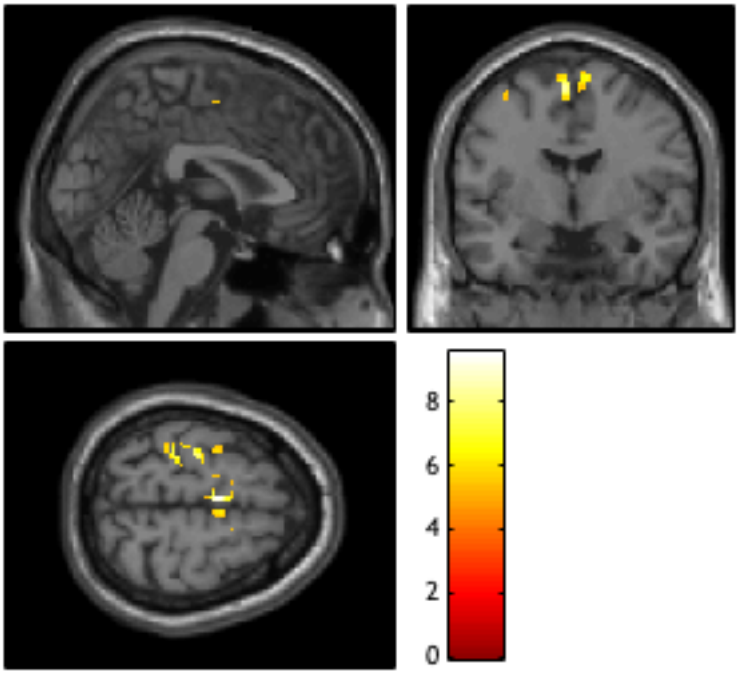


Figure 4.10 A pictorial representation of the cortical activations for forward flexion from the control group, pFWE-corr 0.05.

Table 4.5 Table to show the cortical activations for forward flexion from the control group, pFWE-corr 0.05.

Cluster-Level		Peak		MNI mm			Brodmann's Area
p(FWE-corr)	Activated Voxels	p(FWE-corr)	T	x	y	z	
<0.001	176	<0.001	9.51	-4	-8	64	6
<0.001	252	<0.001	8.21	-28	-20	62	6
<0.001	175	<0.001	7.68	-44	-28	18	13
0.002	27	<0.001	6.75	50	-32	20	13
<0.001	60	<0.001	6.74	8	-4	70	6
0.001	41	0.001	6.39	-6	2	42	24
0.004	18	0.001	6.32	16	-20	40	24
0.001	31	0.005	5.89	-18	-18	18	Ventral Lateral Nucleus

4.2.2.3 Abduction

Table 4.6 and Figure 4.11 illustrate that overall there are 20 clusters generated in the control group during abduction. If the analysis is restricted to 10 or more voxel clusters, then number of clusters reduces to 7. The patient group has a greater number of clusters,

namely 30, and at a voxel threshold of 10, the number of clusters is 8.

In summary, overall there is a greater number of clusters and level of activation in the patient group for the movement of abduction.

Considering the entire table for the control group, without a voxel threshold, the average is 38.7 voxels (SD 81.53), the range is 305 and the total activation is 305. When the threshold is raised to 10 voxels, the mean rises to 104.7 (SD 114.97), the range is 294 and the total activation is 294.

Comparing this with the patient group (Table 4.3), considering all the activations, the mean was 72.7 (SD 211.35), the range is 1017, and the total activation is 2181. If a voxel threshold of 10 is adopted, then the mean activation is 266.25 (SD 355.91), the range 955 and the total activation is 2130.

Table 4.7 illustrates the principal differences between the patient and the control group. For all movement, the patients had the greatest level of activation whereas the controls had a greater number of clusters. Comparing the two groups for the movement of forward flexion, the controls have a comparatively greater level of activation and number of clusters. The situation for abduction is

reversed compared to forward flexion, with the patients having comparatively high levels of activation and number of clusters.

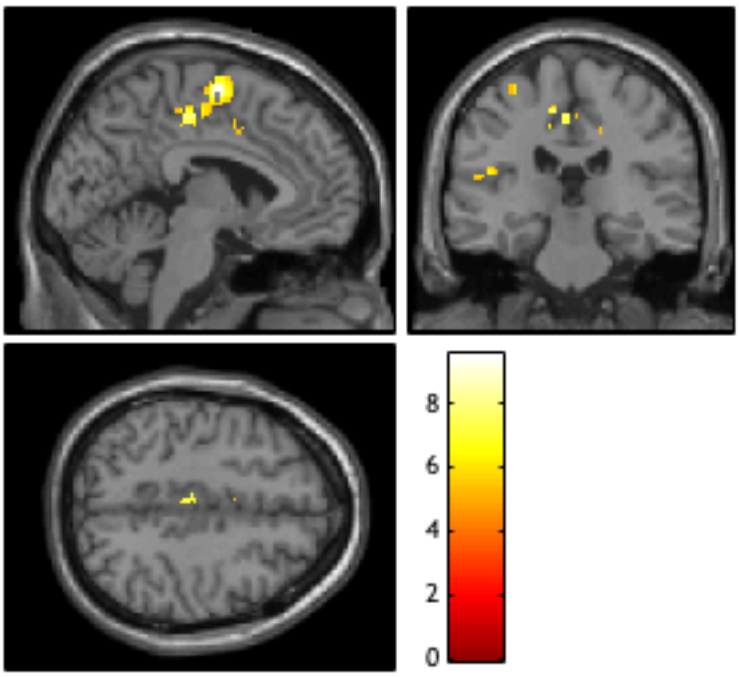


Figure 4.11 A pictorial representation of the cortical activations for abduction from the control group, pFWE-corr 0.05.

Table 4.6 Table to show the cortical activations for abduction from the control group, pFWE-corr 0.05.

Cluster-Level		Peak		MNI			Brodmann's Area
p(FWE-corr)	Activated Voxels	p(FWE-corr)	T	x	y	z	
<0.001	227	<0.001	9.48	-4	-8	64	6
<0.001	306	<0.001	8.15	-28	-20	62	6
<0.001	60	<0.001	6.89	8	-6	70	6
0.001	46	0.001	6.55	-48	-30	20	13
<0.001	70	0.001	6.36	-18	-22	12	Lateral Posterior Nucleus
0.008	12	0.01	5.65	-6	2	42	24

Table 4.7 A summary of the level and number of activations in both the patient and control group (yellow indicating the high value)

	Patients				Controls			
Type	Cluster Number <10 voxels	Cluster Number >10 voxels	Level of Activation <10 voxels	Level of activation >10 voxels	Cluster Number <10 voxels	Cluster Number >10 voxels	Level of Activation <10 voxels	Level of activation >10 voxels
All	30	11	3,763	3,823	36	17	3,259	3,192
Forward Flexion	13	7	393	225	21	10	811	780
Abduction	30	8	2181	2130	20	7	305	294

4.2.2.4 Abduction subtracted from Forward Flexion

As for the patient group no voxels survived the Family Wise Correction when subtracting abduction from forward flexion. This is consistent with the method development study data, Chapter 3.1.5. It was thought that an increase in the number of scans may improve the survival of voxels after Family Wise Correction, particularly in a control group, where it was thought that the activation patterns would be more consistent and less diffuse.

4.2.3 Comparison of Patients versus Controls

When the activations of the control group (n=16) were subtracted from the patient group (n=16) at a voxel level result is illustrated in Table 4.8, Figures 4.13 and 4.14. The MNI co-ordinates for the 2 mm x 2 mm x 2 mm voxel are -38 -26 56. Figure 4.15 shows the MNI co-ordinates within the cluster activation, demonstrating the location within the left post central gyrus, which is the location of the primary somatosensory cortex of the parietal lobe, Figure 4.16.

The somatosensory cortex includes Brodmann areas 1, 2 and 3. All these Brodmann areas are receptive areas for the sense of touch. Figure 4.17 shows the output from the SPM Anatomy Probability Atlas) [310-314], showing a probability of 52% (33-63%) of being within Brodmann area 4a [318], probability of 37% (17-43%) for Brodmann area 4p and probability of 11% (7-28%) for Brodmann area 3b. The implications of these results will be discussed in detail in the discussion Chapter 6.

The computation was repeated for all movement of the controls subtracted from the patients at the FWE cluster level. The results of this further analysis are illustrated at Table 4.9. This shows surviving activations at pFWE-corr 0.001 (as recommended by Woo et al.[271]), showing five areas of cluster within postcentral gyrus,

anterior division of the supramarginal gyrus, pars opercularis within inferior frontal gyrus, and two areas within the precentral gyrus.

Table 4.8 demonstrates that for the areas within the precentral and supramarginal gyrus the MNI coordinates fell firmly within the Brodmann areas 6, 40 and 44 using the WFU_Pickaltas [315, 316]. As the clusters were relatively large (between 430 and 769 clusters), SPM Anatomy Toolbox (Version 2.1), [310-314] was not appropriate as the areas could include more than one Brodmann area. Using the -28 coronal slices, as a reference slice the boundaries were explored, and this section shows the cluster inclusive of the coordinates from the voxel surviving FWE at a voxel level. The cluster at the lateral edge (Figure 4.19) fell within Brodmann area 3. The cluster at the superior edge is inferior and medial (Figure 4.20, 4.21 and 4.22) fell within Brodmann area 4.

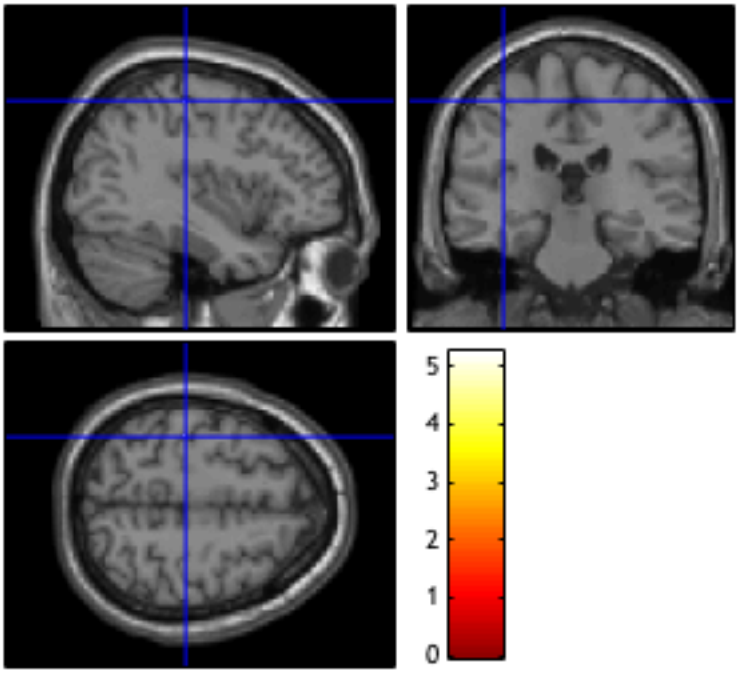


Figure 4.13 A pictorial representation of the cortical activations for all movement of controls subtracted from the patient group showing the MNI co-ordinates of -38, -26, 56, pFWE-corr 0.05.

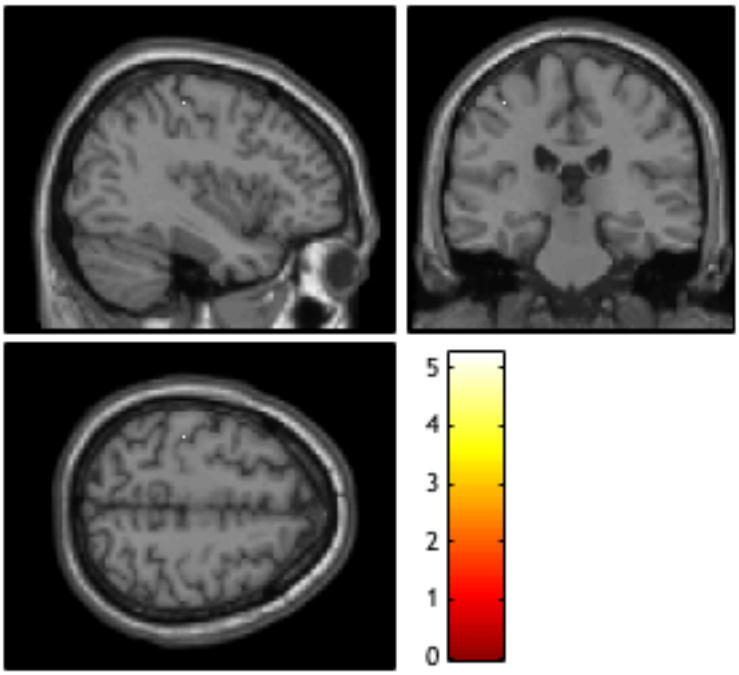


Figure 4.14 A pictorial representation of the cortical activations for all movement of controls subtracted from the patient group showing the activation at the voxel level, MNI co-ordinates -38, -26 56, pFWE-corr 0.05.

Table 4.8 Table to show the cortical activations for all movement of controls subtracted from the patient group at a voxel level, pFWE-corr 0.05.

Cluster p(FWEcorr)	Voxels	Peak p(FWEcorr)	Peak T value	MNI Coordinates		
				x	y	z
0.035	1	0.04	5.22	-38	-26	56

Figure 4.15 A graphical representation of the cortical activations for all movement of controls subtracted from the patient group showing the activation level at a cluster level at the MNI location -38, -26, 56, pFWE-corr 0.001.

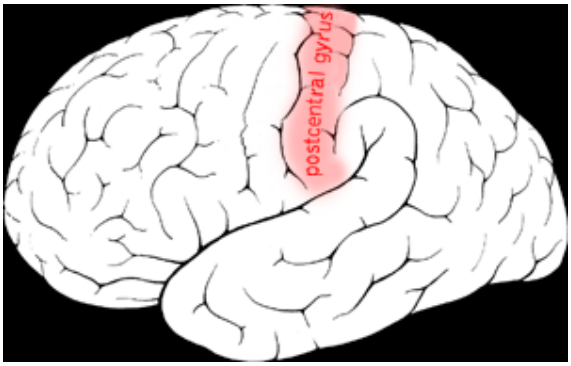


Figure 4.16 Representation of the postcentral gyrus of the parietal lobe.

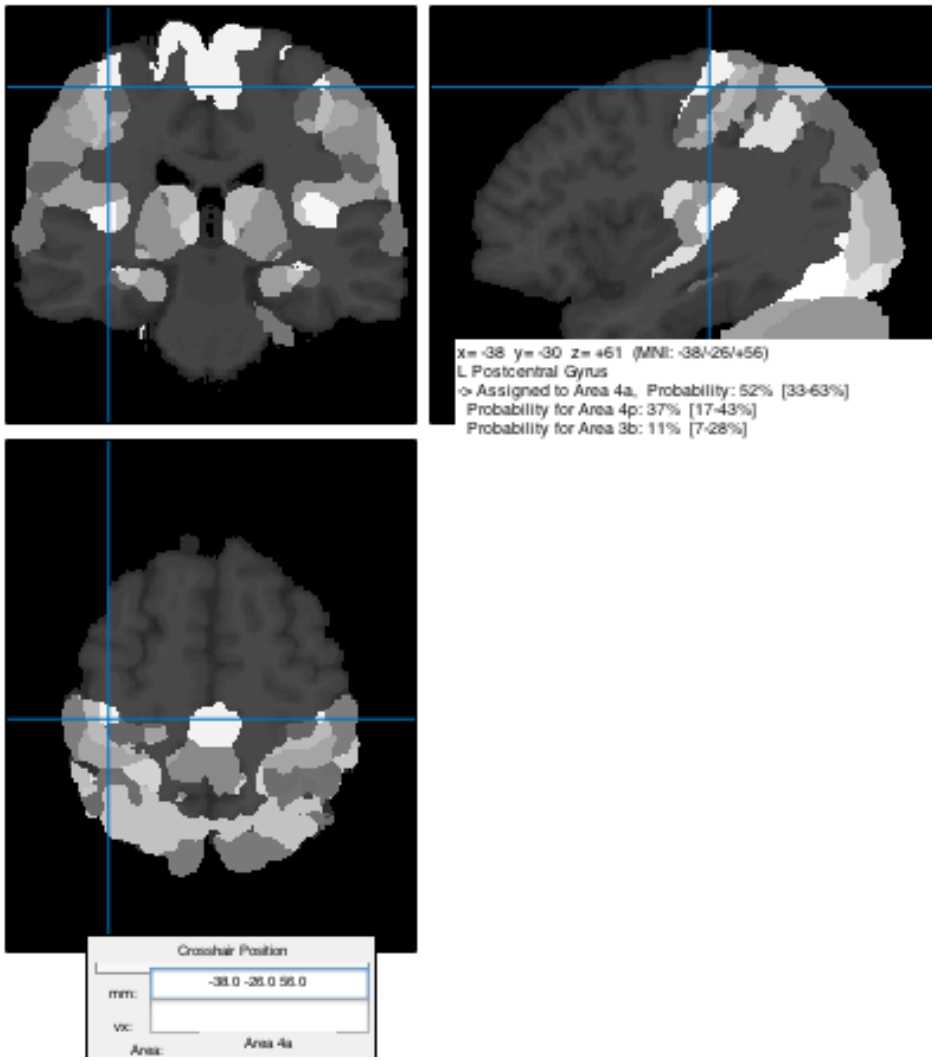


Figure 4.17 This shows the results from Anatomy Probability SPM atlas) [310-314] of the MNI coordinates -38 -26 56.

Table 4.9 Table to show the cortical activations for all movement of controls subtracted from the patient group at a cluster level, pFWE-corr 0.001.

Region	Voxel Size	T value	MNI coordinates			Brodmann Area from WFU_Pickaltas	Brodmann Area from Cluster Exploration
			x	y	z		
Postcentral Gyrus	430	5.22	-38	-26	56		3,4
Supramarginal Gyrus anterior division	430	4.24	-56	-36	44	40	
Inferior Frontal Gyrus pars opercularis	769	4.87	-44	12	22	44	9
Precentral Gyrus	769	4.2164	-40	-2	52	6	

4.2.3.1 Postcentral Gyrus

Figure 4.18, 4.19, 4.20, 4.21 and 4.22, with Table 4.9 demonstrate this cluster consisted of 430 voxels, with a T value of 5.2. The method of locating the Brodmann areas 3 and 4 within this cluster has been described in the previous section. (NB: Brodmann area 4 is the primary motor cortex located within the precentral gyrus and Brodmann area 3 is part of the primary somatosensory cortex within the postcentral gyrus.)

Figure 4.18 A pictorial representation of the cortical activations for all movement of controls subtracted from the patient group showing the activation level at a cluster level at the MNI location -38 -26 56, pFWE-corr 0.001.

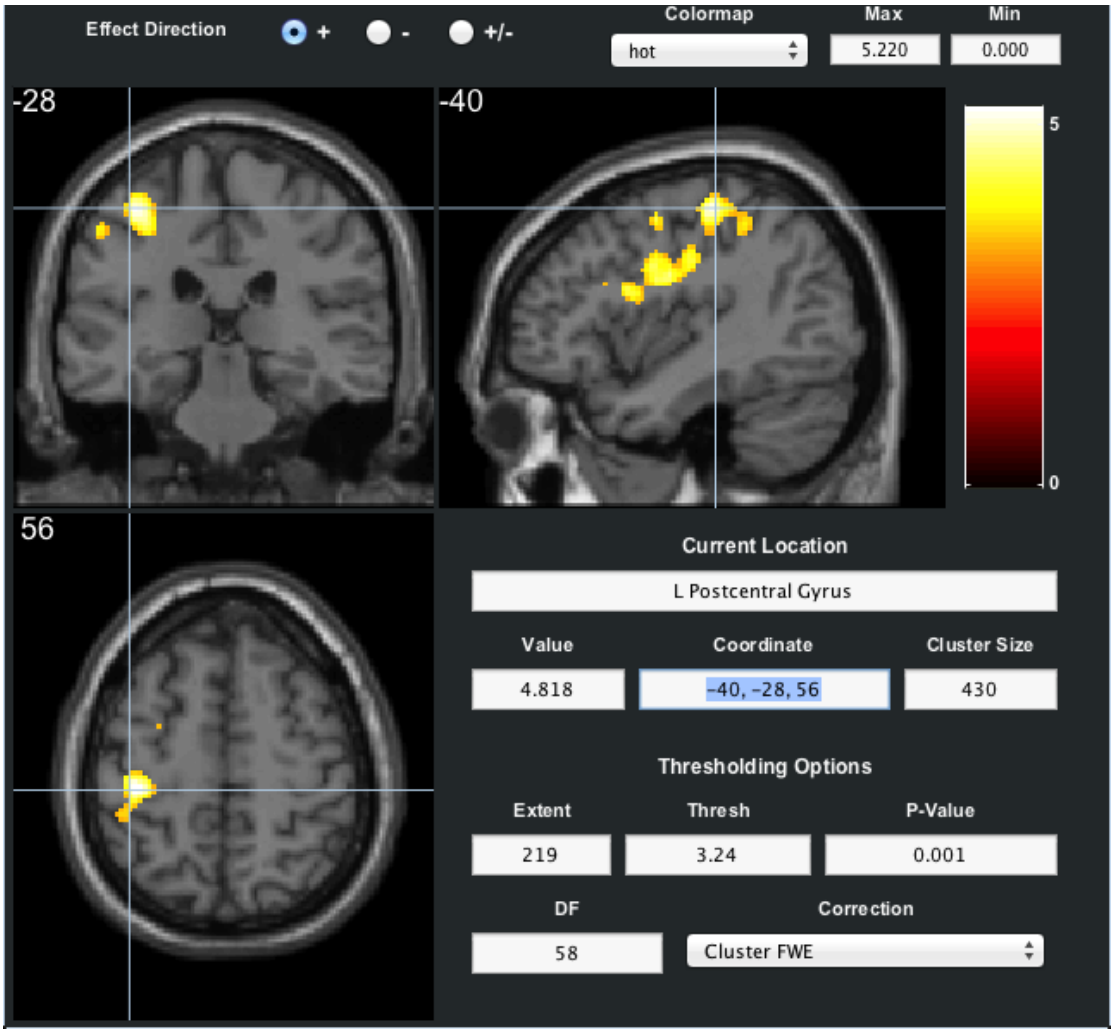


Figure 4.19 A pictorial representation of the cortical activations within Brodmann's area 3 at MNI location -40, -28, 56 for all movement of controls subtracted from the patient group showing the activation level at a cluster level, pFWE-corr 0.001.

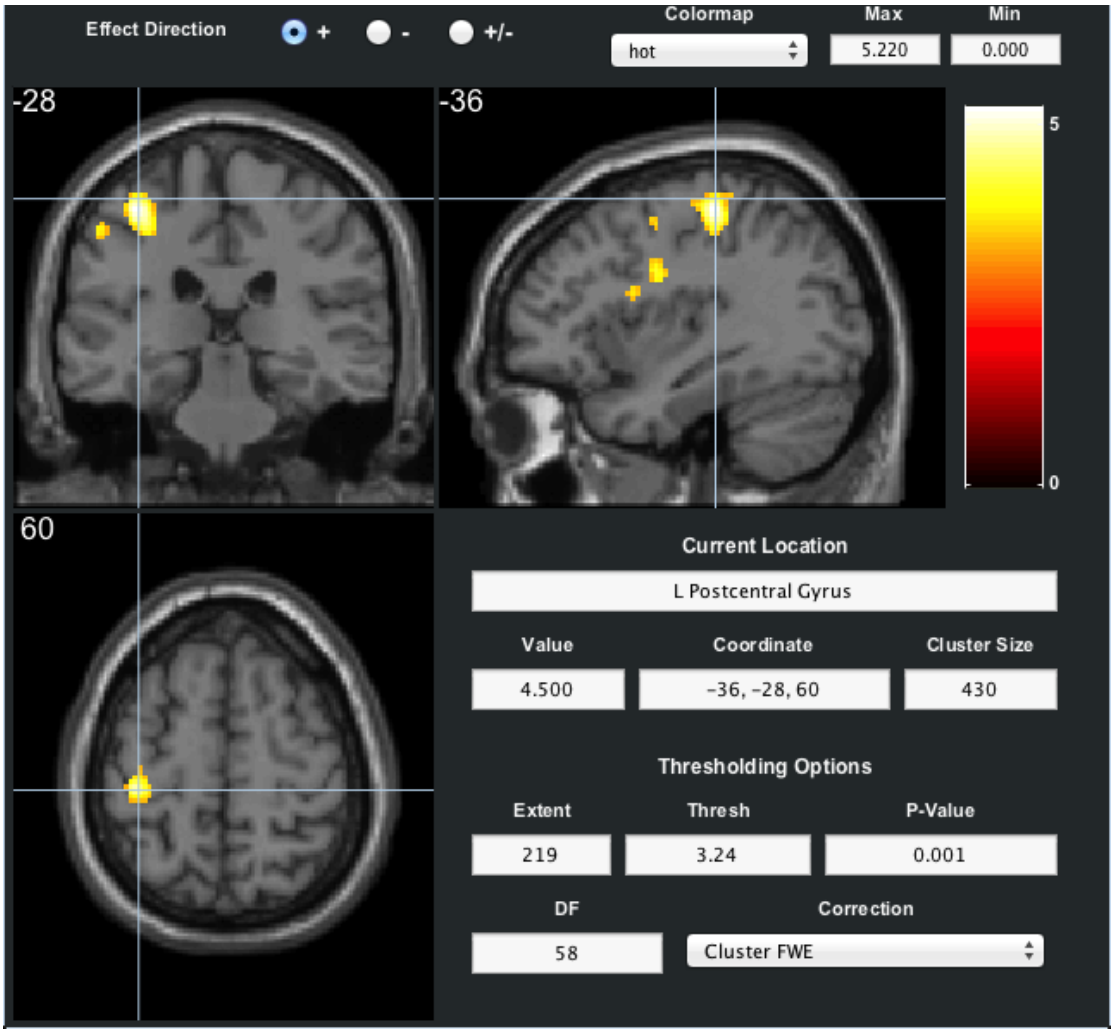


Figure 4.20 A pictorial representation of the cortical activations within Brodmann area 4 at MNI location -36, -28, 60 for all movement of controls subtracted from the patient group showing the activation level at a cluster level, pFWE-corr 0.001.

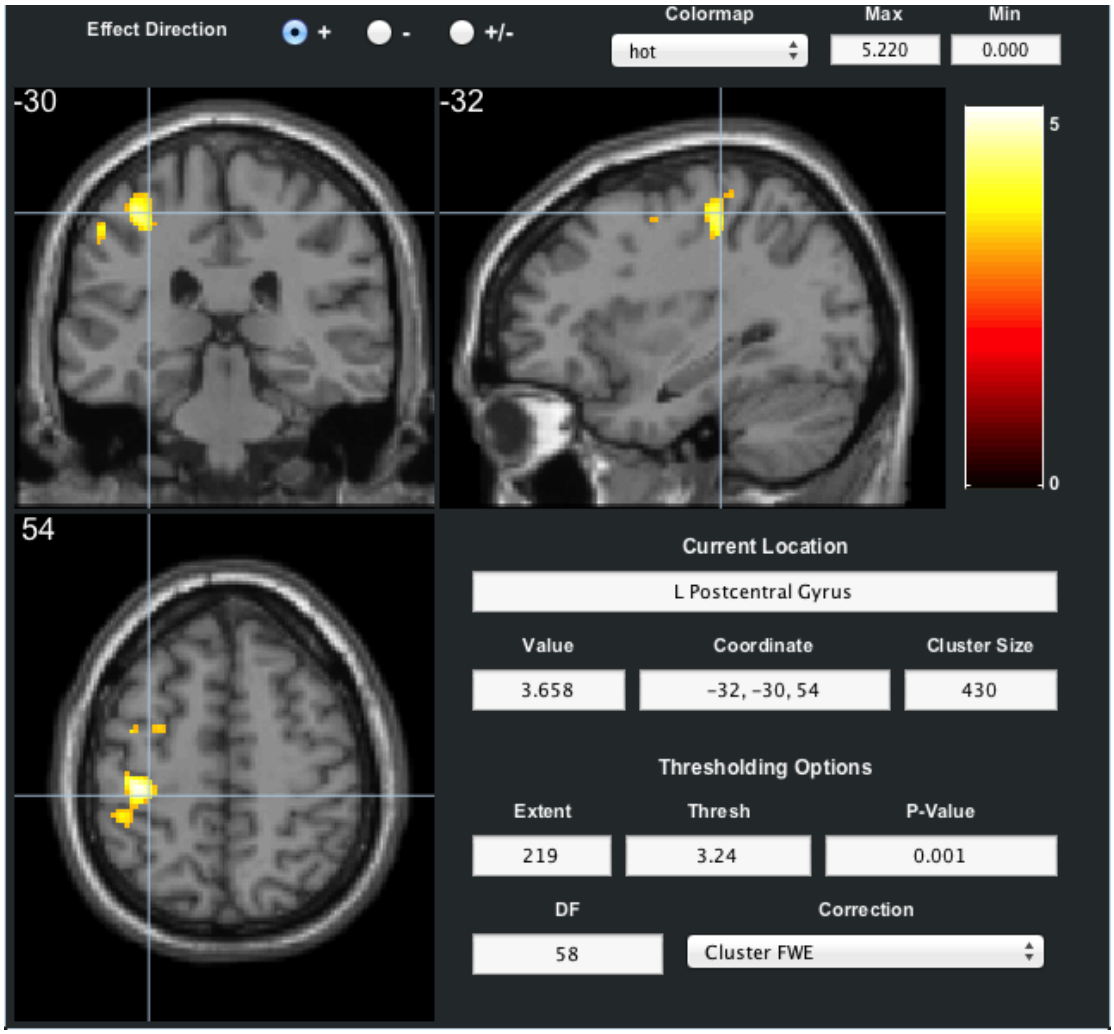


Figure 4.21 A pictorial representation of the cortical activations within Brodmann area 4 at MNI location -32, -20, 54 for all movement of controls subtracted from the patient group showing the activation level at a cluster level, pFWE-corr 0.001.

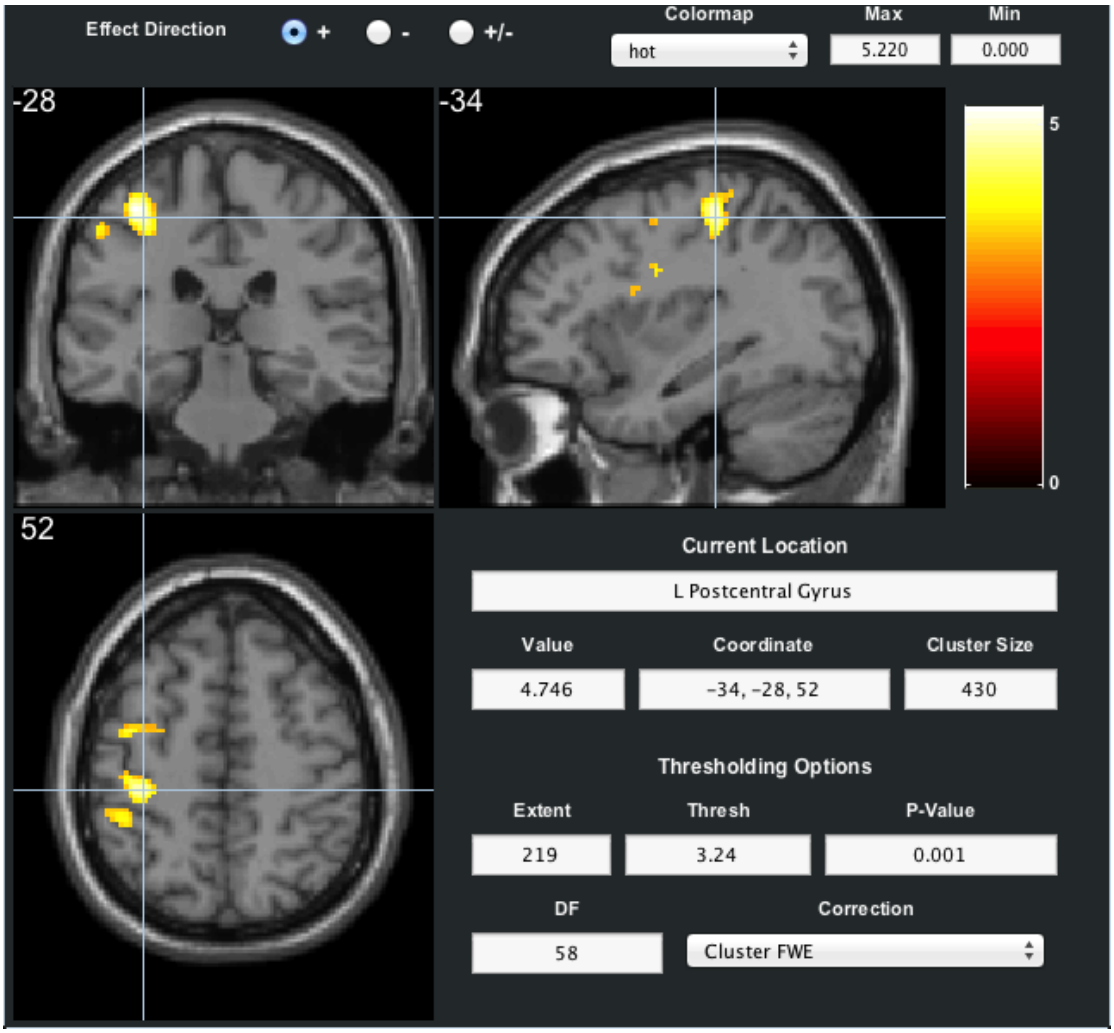


Figure 4.22 A pictorial representation of the cortical activations within Brodmann area 4 at MNI location -34, -28, 52 for all movement of controls subtracted from the patient group showing the activation level at a cluster level, pFWE-corr 0.001.

4.2.3.2 Supramarginal Gyrus anterior division

The cluster consisted of 430 voxels, with a T value of 4.24 located within the parietal cortex, which functions at a simplistic level and relates to reading in terms of meaning and phonology [319].

Figure 4.23 A pictorial representation of the cortical activations for all movement of controls subtracted from the patient group showing the activation level at a cluster level at the MNI location -56 -36 44, pFWE-corr 0.001.

4.2.3.3 Inferior Frontal Gyrus pars opercularis

Figure 4.24 shows the peak cluster coordinates (Table 4.9); the extent of the activation was 769 voxels, which was greater than clusters set out in the previous two sections. The t value for the cluster was 4.87.

The peak of the cluster coordinates did not show within a Brodmann area. The pars opercularis of the inferior frontal gyrus is associated with three Brodmann areas, 6, 9 and 44. A systematic exploration of the cluster was examined and the MNI coordinates -46 4 36 showed as areas within Brodmann area 9.

Figure 4.24 A pictorial representation of the cortical activations for all movement of controls subtracted from the patient group showing the activation level at a cluster level at the MNI location -44 12 22, pFWE-corr 0.001.

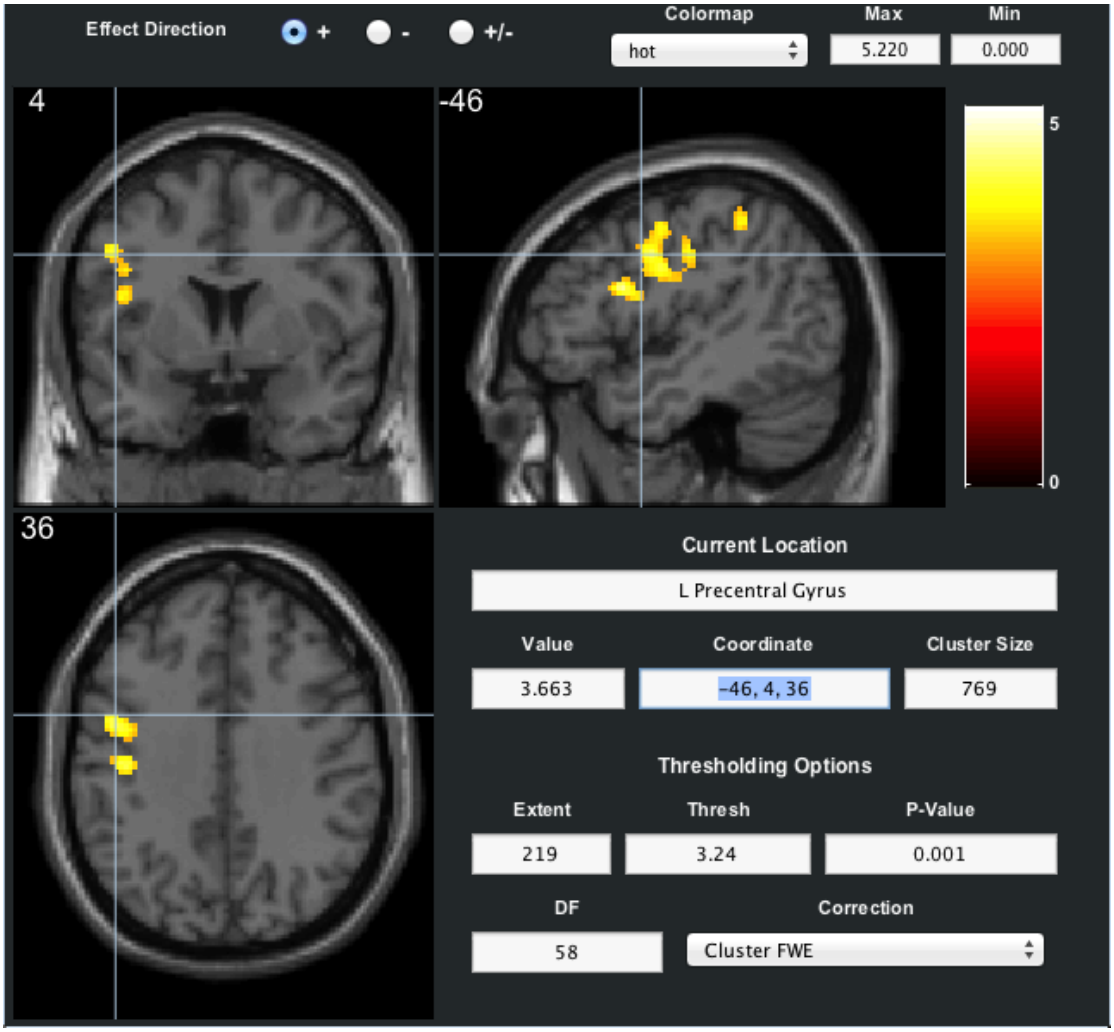


Figure 4.25 A pictorial representation of the cortical activations within Brodmann area 9 at MNI location -46, 4, 36 for all movement of controls subtracted from the patient group showing the activation level at a cluster level, pFWE-corr 0.001.

4.2.3.4 Precentral Gyrus

Figure 4.26 illustrates two clusters within Brodmann area 44 (Table 4.9), the extent of the cluster is 769 voxels and the T value is 4.54.

The second cluster in this region is a cluster within Brodmann area 6, shown at Figure 4.27. The extent of the cluster was identical to the other cluster is this gyrus, at 769 voxels and there was a comparable t value of 4.2.

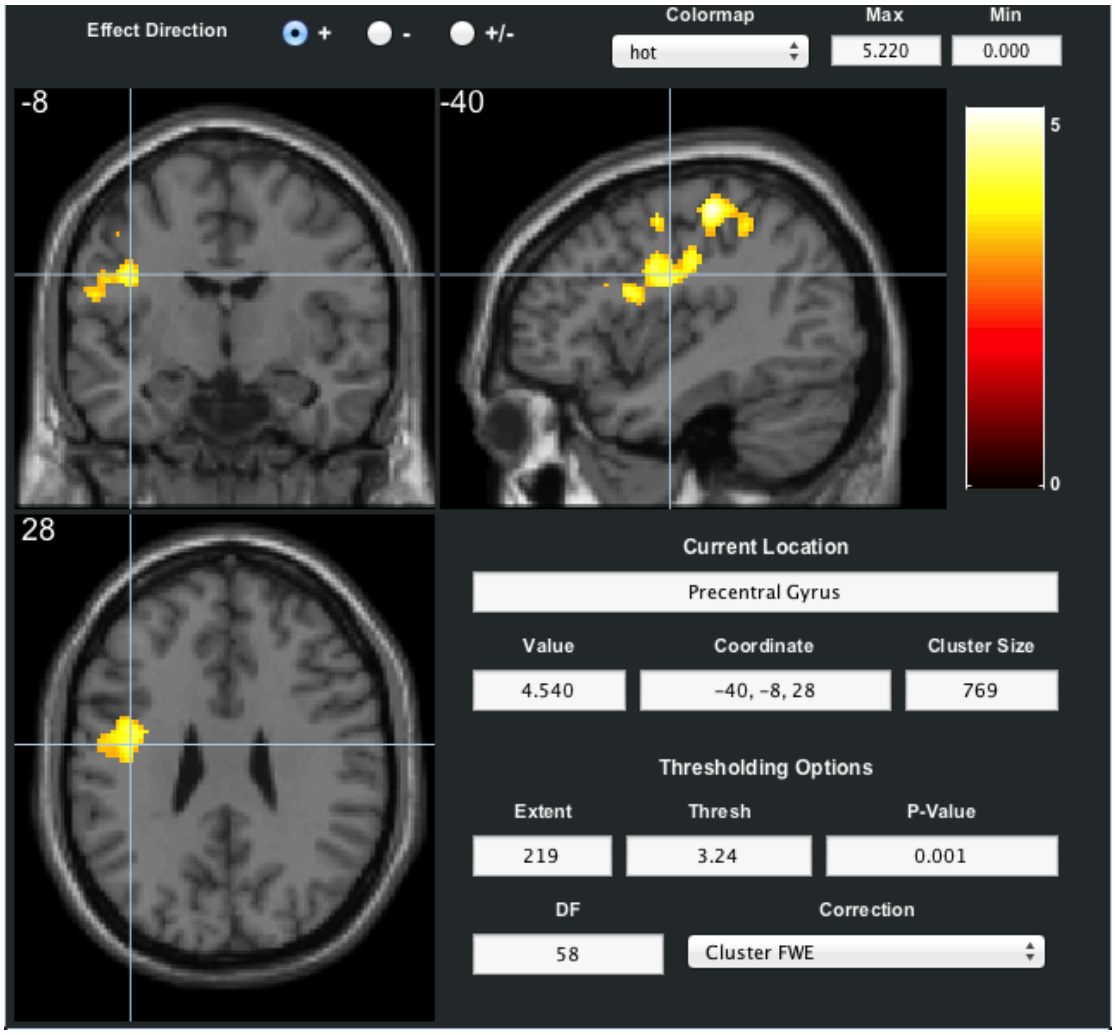


Figure 4.26 A pictorial representation of the cortical activations for all movement of controls subtracted from the patient group showing the activation level at a cluster level at the MNI location -40 -8 28, Brodmann area 44, pFWE-corr 0.001.

Figure 4.27 A pictorial representation of the cortical activations for all movement of controls subtracted from the patient group showing the activation level at a cluster level at the MNI location -40, -2, 52, Brodmann area 6, pFWE-corr 0.001.

4.2.3.5 Comparison of Brodmann Areas

4.2.3.5.a No Minimum Threshold of voxels

An analysis was undertaken to consider the differences in the location of the voxel activations surviving the Family Wise Correction at a voxel level. Brodmann areas were taken from the WFU_picklas.

A summary of the Brodmann areas present and absent comparing the two groups is set out in Table 4.10.

Figure 4.28 illustrates the Brodmann areas activated in both the patient and control group in all movement (forward flexion and abduction) without any threshold voxel applied. Activation is present in patient group but absent in the control group in Brodmann areas 24, 39, 42 and VLN. It is present in the control group but absent from the patient group in Brodmann areas 7, 10 and 37.

For the movement of forward flexion, Figure 4.29, activation in Brodmann areas 4, 31, 35 and 40 is present in the patient group compared to the control group. Activation in Brodmann areas 19, 41, Putamen and VLN is present in the control group but absent in the patient group.

When the analysis is undertaken for abduction (Figure 4.30), activation in Brodmann areas 4, 5, 40 and the corpus callosum are present in the patient group but absent in the control group. Further, the Putamen and the lateral posterior are present in the control group only.

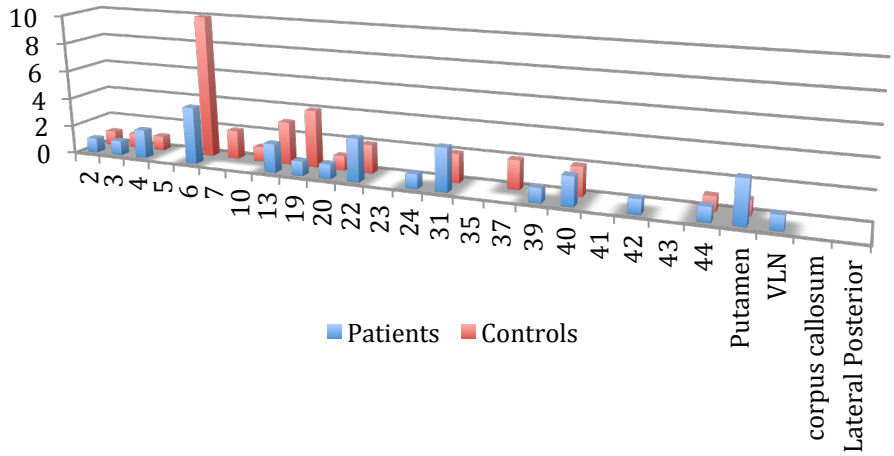


Figure 4.28 Bar graph to show the different Brodmann area activations comparing patients (blue) against controls (red) for all movement (forward flexion and abduction), recording all activations clusters with no minimum threshold, pFWE=0.05

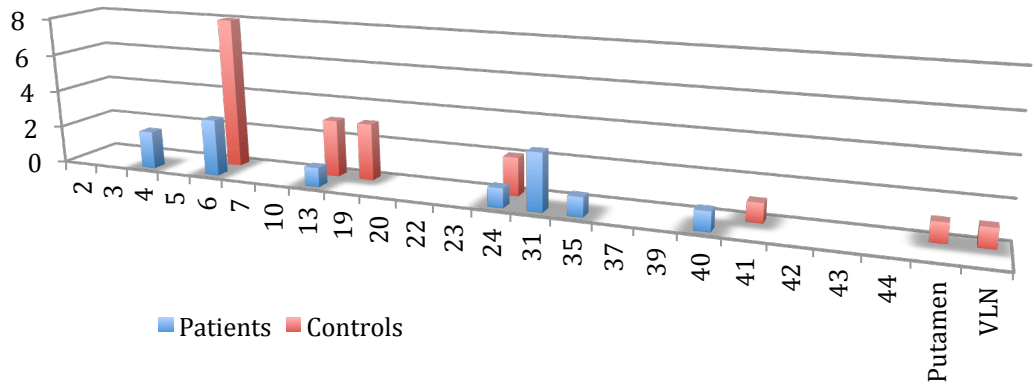


Figure 4.29 Bar graph to show the different Brodmann's area activations comparing patients (blue) against controls (red) for forward flexion, recording all activations clusters with no minimum threshold, pFWE=0.05

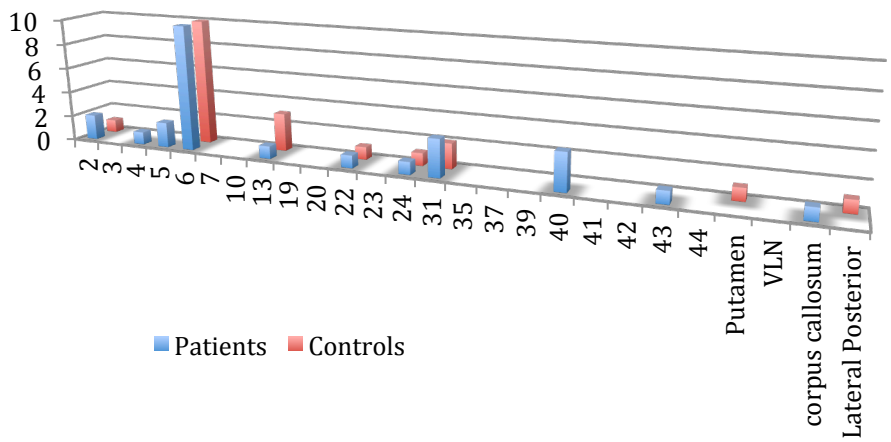


Figure 4.30 Bar graph to show the different Brodmann area activations comparing patients (blue) against controls (red) for abduction, recording all activations clusters with no minimum threshold, pFWE=0.05

4.2.3.5.b Minimum Threshold of 10 voxels

Table 4.10 illustrates the Brodmann areas present and absent comparing the two groups in summary format.

As illustrated in Figure 4.31 Brodmann areas activated in both the patient and control group in all movement (forward flexion and abduction) with a threshold of 10 voxels applied. Activation present in the patient group but absent in the control group is shown in Brodmann areas 4, 42 in addition to Putamen and VLN. Activation present in the control group but absent in the patient group is shown in Brodmann areas 6, 10, 13, 37, Corpus Callosum.

For the movement of forward flexion, Figure 4.32, activation is present in Brodmann areas 4, and 31 in the patient group compared to the control group, while in Brodmann areas 23 and VLN activation is present in the control group but absent in the patient group.

For the movement of abduction as Figure 4.33 illustrates, activation is present in Brodmann areas 4, 22, 31 and 43 in the patient group but absent in the control group, while the Putamen and the lateral posterior activation are present in the control group only.

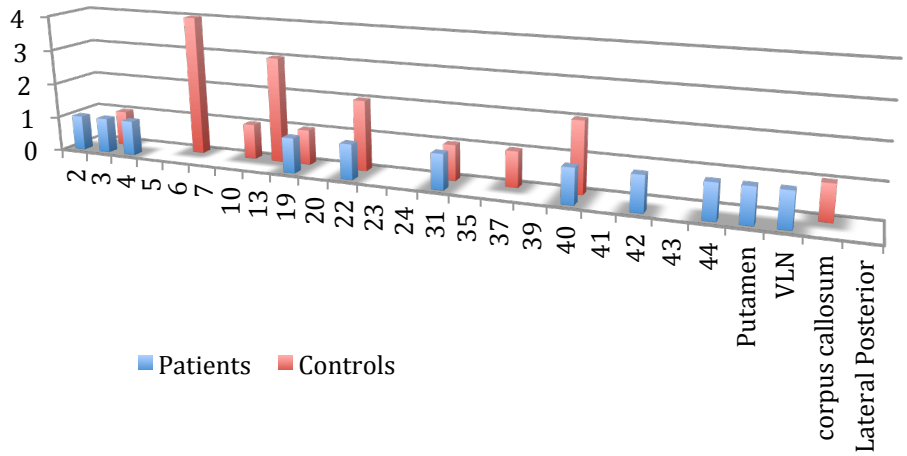


Figure 4.31 Bar graph to show the different Brodmann area activations comparing patients (blue) against controls (red) for all movement (forward flexion and abduction), recording all activations clusters with a minimum 10 voxel threshold, pFWE=0.05

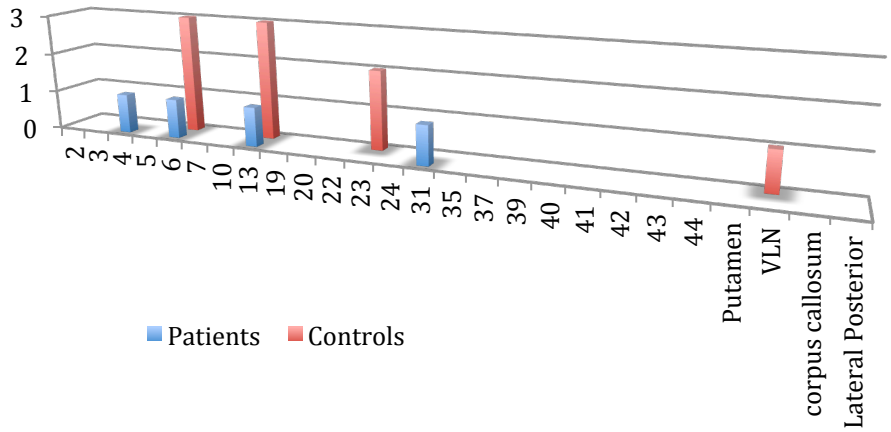


Figure 4.32 Bar graph to show the different Brodmann area activations comparing patients (blue) against controls (red) for forward flexion, recording all activations clusters with a minimum 10 voxel threshold, pFWE=0.05

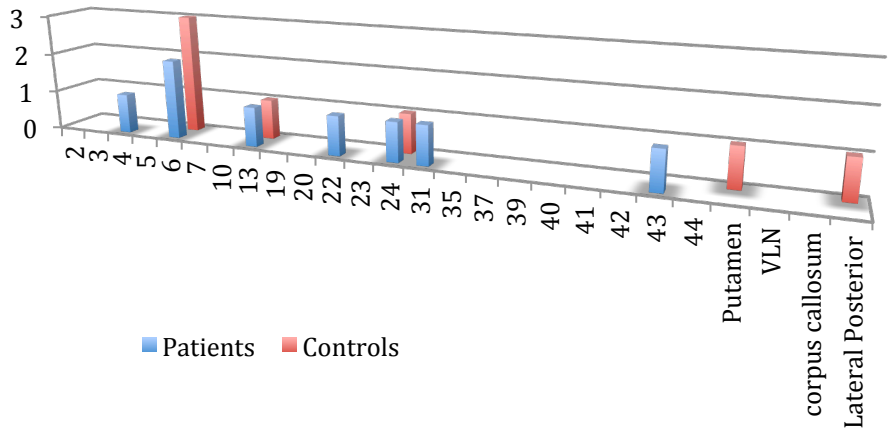


Figure 4.33 Bar graph to show the different Brodmann area activations comparing patients (blue) against controls (red) for abduction, recording all activations clusters with a minimum 10 voxel threshold, pFWE=0.05

Table 4.10 A summary of the Brodmann areas which are present and absent over during the three types of movement.

	Patients				Controls			
Type	Comparative Brodmann areas present		Comparative Brodmann areas absent		Comparative Brodmann areas present		Comparative Brodmann areas absent	
	<10 Voxels	>10 Voxels	<10 Voxels	>10 Voxels	<10 Voxels	>10 Voxels	<10 Voxels	>10 Voxels
All	24, 39, 42, VLN	4, 42, Putamen VLN	7, 10, 37	6, 10, 13, 37 Corpus Callosum	7, 10, 37	6, 10, 13, 37 Corpus Callosum	24, 39, 42, VLN	4, 42, Putamen VLN
Forward Flexion	4, 31, 35, 40	4, 31	19, 41, Putamen, VLN	23 VLN	19, 41, Putamen, VLN	23 VLN	4, 31, 35, 40	4, 31
Abduction	4, 5, 40 corpus callosum	4, 22, 31, 43	Putamen Lateral Posterior	Putamen Lateral Posterior	Putamen Lateral Posterior	Putamen Lateral Posterior	4, 5, 40 corpus callosum	4, 22, 31, 43

4.2.4 First Level retrospective Analysis

As set out in Chapter 4.2.3, a single voxel survived the Family Wise Correction at the voxel level at MNI coordinates -38 -26 56 (Figure 4.13 and 4.14). The patient group demonstrated a great variance in WOSSI and OIS scores compared to the controls, but it can be seen that P1 had to all intense and purposes a normal shoulder following treatment.

A first level analysis was repeated in SPM 12 for P1. The 6 motion parameters were modeled as regressors and grey matter mask applied. The model was estimated and a contrast of rest subtracted from all movement was applied. Figure 4.34 illustrates the location of MNI coordinates -38 -26 56 is show by the crosshairs. It can be seen at this location in P1 there is no activation. The same exercise was repeated for P2, illustrated in Figure 4.35 which showed high scores in both the WOSI and the OIS. The exercise was repeated for the others within patient group and the activation was present in all.

Figure 4.34 A pictorial representation of the cortical activations for all movement of at First Level Analysis for P1, showing the activation level at the MNI location -38, -26, 56, pFWE-corr 0.001.

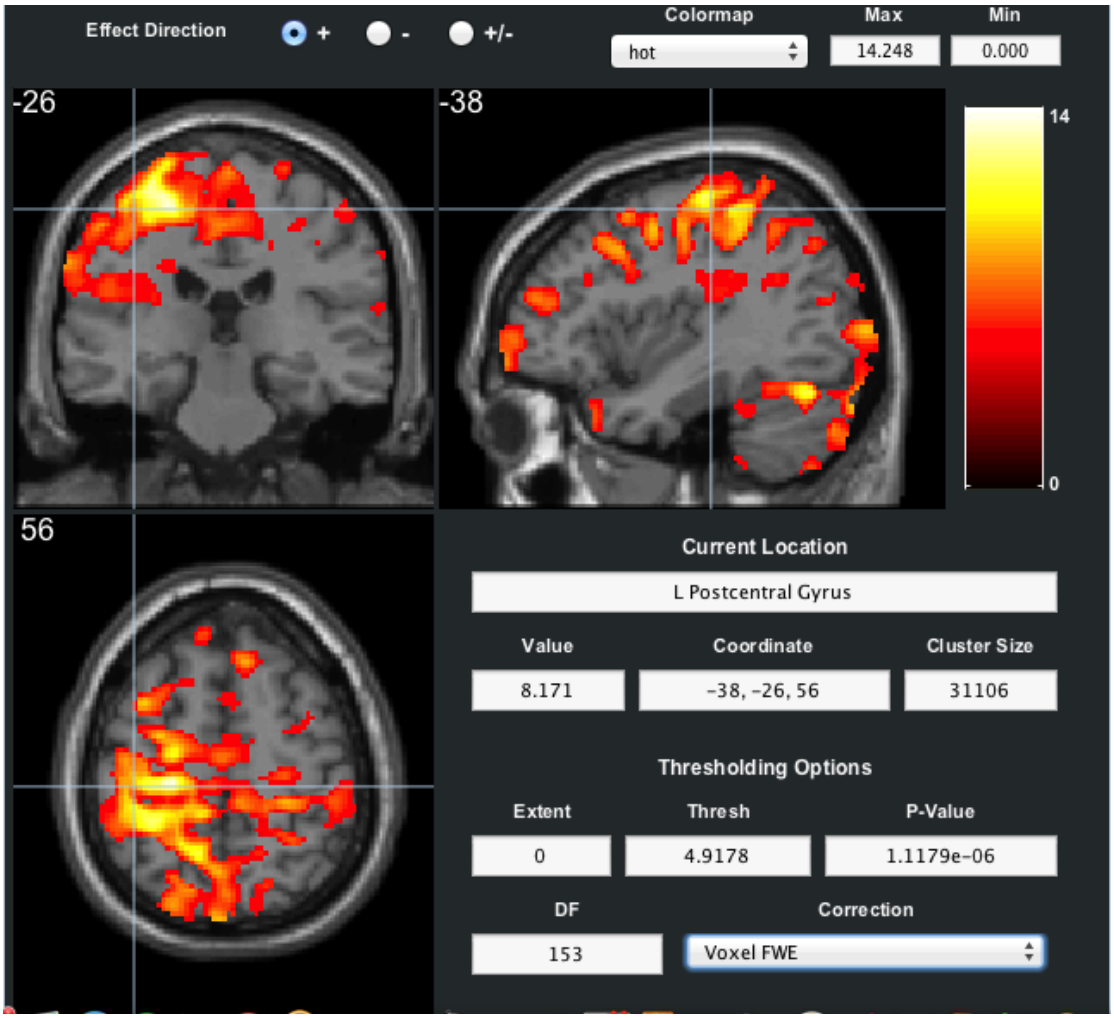


Figure 4.35 A pictorial representation of the cortical activations for all movement of at First Level Analysis for P2, showing the activation level at the MNI location -38, -26, 56, pFWE-corr 0.001.

4.2.5 The Western Ontario Shoulder Instability Index

As illustrated in Figure 4.36 and Table 4.11, when WOSI was used as covariant, there were increased activations in Brodmann areas amyydala, 3, 6, 11 and 26.

The amgydala is part of the limbic and located within the temporal lobe (note that Brodmann area 3 is the primary somatosensory cortex, Brodmann area 6 is the premotor cortex, Brodmann 11 is orbitofrontal area and Brodmann area 26 ectosplenial)

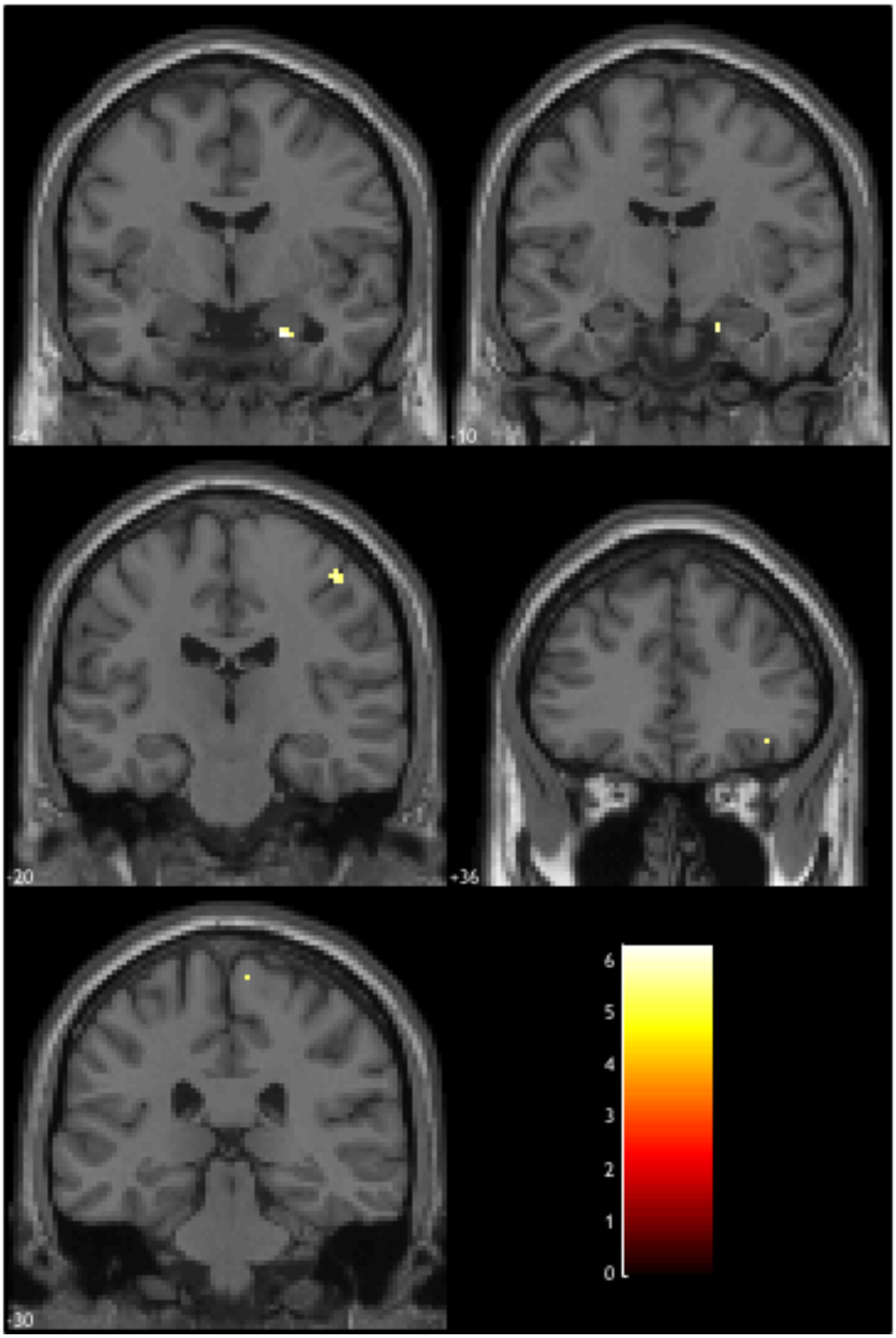


Figure 4.36 A pictorial representation of the cortical activations for all movements (Forward Flexion and Abduction) with the Western Ontario Shoulder Instability Index as a covariate at a voxel level, pFWE-corr 0.05.

Table 4.11 Table to show the cortical activations for all movements (Forward Flexion and Abduction) with the Western Ontario Shoulder Instability Index as a covariate at a voxel level, pFWE-corr 0.05.

Cluster-Level		Peak		MNI			Brodmann's Area
p(FWE-corr)	Activated Voxels	p(FWE-corr)	T	x	y	z	
0.027	9	0.004	6.33	22	-4	-26	amygdala
0.033	5	0.013	5.85	20	-10	-24	26
0.023	13	0.016	5.78	44	-20	56	3
0.043	1	0.027	5.54	40	36	-12	11
0.037	3	0.033	5.46	8	-30	72	6

4.2.6 Oxford Shoulder Instability Score

As illustrated in Figure 4.36 and Table 4.11, when OSIS was used as covariant, there were increased activations in Brodmann area amygdala.

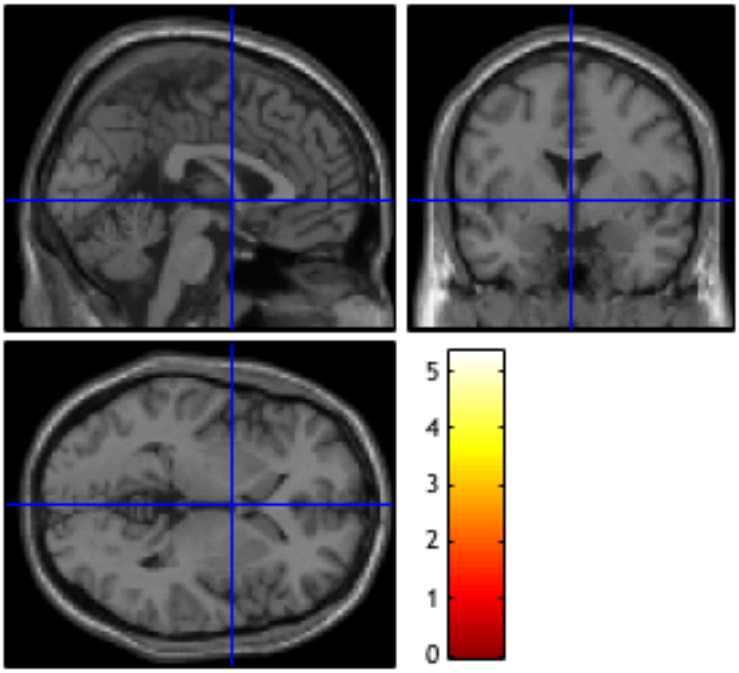


Figure 4.37 A pictorial representation of the cortical activations for all movements (Forward Flexion and Abduction) with the Oxford Shoulder Instability Score at a Covariate as a voxel level, crosshairs showing the MNI co-ordinates of 24 -4 -26, pFWE-corr 0.05.

Table 4.12 Table to show the cortical activations for all movements (Forward Flexion and Abduction) with the Oxford Shoulder Instability Score as a Covariate at a voxel level, pFWE-corr 0.05.

Cluster-Level		Peak		MNI			Brodmann Area
p(FWE-corr)	Activated Voxels	p(FWE-corr)	T	x	y	z	
0.044	1	0.044	5.31	24	-4	-26	Amygdala

5 Electromyography Results

In this chapter I will present the electromyography results, dividing the presentation into the movements of forward flexion/extension and abduction/adduction, starting with a shoulder that has suffered no known pathology and then considering EMG data from patients with Polar type II/III instability.

The graphs showing the detail muscle activation profile for each of the muscles tested is set out in four appendices:

Normal Shoulder Group – Forward Flexion – Appendix 1
Comparative Study – Forward Flexion – Appendix 2
Normal Shoulder Group – Abduction/Adduction – Appendix 3
Comparative Study – Abduction/Adduction – Appendix 4

Throughout the results section, some individual muscles activation graphs will be shown in order illustrate some of the points being made in the narrative.

5.1 Normal Shoulder Group - Forward Flexion

5.1.1 Global Analysis

5.1.1.1 Standing

Full results for the mean amplitude for the 12 individual muscles are reported in Table 5.1, and graphically illustrated in Figure 5.1. Set out in Chapter 3.2.4, shows Phase 1 representing the upstroke from 0 to 180 degrees, and Phase 2 representing the reverse movement. It can be seen that there are significant differences ($p=<0.001$ to $p=0.042$) between the phases. Further, as one might expect, greater activation occurs in Phase 1 compared to Phase 2; this is particularly pronounced with AD, MD, the primary flexors and ISP and SUB of the posterior cuff.

The mean amplitude is between 108.7-124.4% (range 15.7) during Phase 1, which is lower in Phase 2, 66.7-84% (range 17.3).

*Table 5.1. Table reporting mean signal amplitude of the normal shoulder group during the movement of forward flexion whilst standing during phase 1 (upward vertical movement from 0 to 180 degrees) and phase 2 (down movement from 180 to 0 degrees) of 13 muscles. SEM is the standard error of measurement. *The t-test assessed whether there was a difference between the two phases.*

		Phase 1			Phase 2		
Muscles	n	Mean (%)	SEM	n	Mean (%)	SEM	t test *
AD	19	124.4	7.3	19	69.1	6.2	0.000
MD	18	127.0	6.6	18	66.7	6.1	0.000
PD	18	110.3	6.7	18	82.3	5.7	0.018
UT	17	117.0	7.1	17	73.5	5.7	0.000
SA	16	112.7	7.5	16	78.0	6.0	0.007
TM	13	109.2	8.7	13	83.0	9.6	0.001
LD	17	104.5	7.0	17	84.0	6.0	0.008
PM	11	120.6	6.2	11	78.4	5.4	0.004
BB	13	116.9	8.9	13	70.5	6.7	0.001
SSP	17	108.7	6.4	17	79.6	5.2	0.000
ISP	13	123.3	7.6	13	77.4	7.0	0.014
SUB Scap	8	127.8	9.7	8	74.7	8.9	0.042

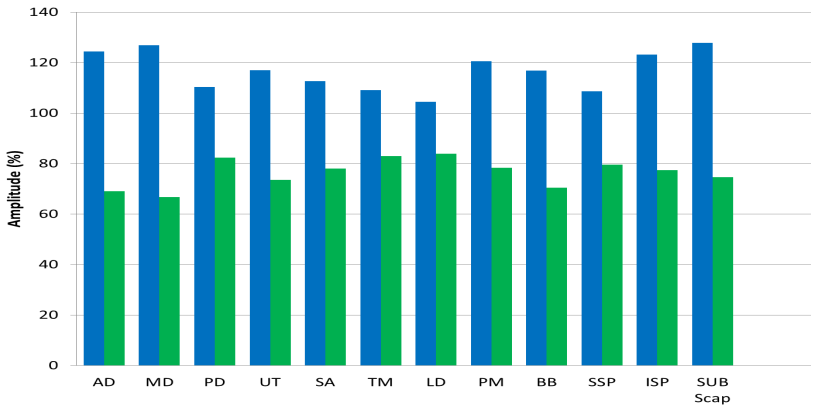


Figure 5.1. Graph of *mean signal amplitude of the normal shoulder group during the movement of forward flexion whilst standing during phase 1 (Blue), (upward vertical movement from 0 to 180 degrees), and phase 2 (Green), (down movement from 180 to 0 degrees) of 13 muscles.*

5.1.1.2 Supine

Compared to the standing forward flexion, although the maximum amplitude is slightly lower (Table 5.2, Figure 5.2) the range of movement is smaller, 30-40 degrees compared to the 180 degrees.

As with the standing position, there is a significant difference in muscle amplitude between Phase 1 and Phase 2.

The four muscles with the greatest amplitude are AD, MD, ISP and UT, which is identified for the standing position of this movement.

*Table 5.2. Table reporting mean signal amplitude of the normal shoulder group during the movement of forward flexion whilst supine during phase 1 (upward vertical movement from 0 to 180 degrees) and phase 2, (down movement from 180 to 0 degrees) of 13 muscles. SEM is the standard error of measurement. *The t-test assessed whether there was a difference between the two phases.*

	Phase 1			Phase 2			
Muscles	n	Mean (%)	SEM	n	Mean (%)	SEM	t test *
AD	22	118.1	4.5	22	72.1	3.1	0.000
MD	24	114.1	3.7	24	80.9	3.5	0.000
PD	23	108.6	3.3	23	87.6	3.3	0.002
UT	16	113.0	3.2	16	79.8	2.8	0.000
SA	16	108.3	3.1	16	85.6	2.7	0.000
TM	19	109.0	2.8	19	87.4	2.8	0.000
LD	22	102.9	2.1	22	91.8	2.3	0.001
PM	23	111.0	2.6	23	83.4	2.5	0.000
BB	14	111.3	3.4	14	83.7	2.8	0.000
SSP	21	107.9	3.1	21	85.4	3.1	0.000
ISP	22	113.3	3.8	22	76.2	3.4	0.000
SUB Scap	14	110.3	4.2	14	81.8	3.4	0.001

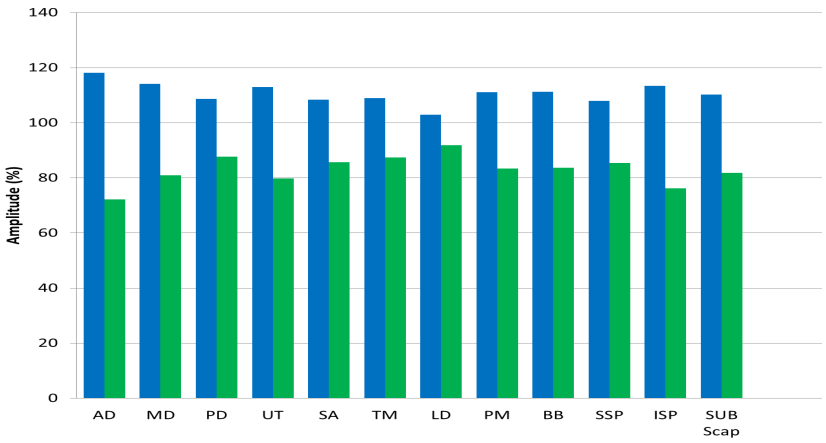


Figure 5.2. Graph to show *mean signal amplitude of the normal shoulder group during the movement of forward flexion whilst supine during phase 1 (Blue) (upward vertical movement from 0 to 180 degrees), and phase 2 (Green) (down movement from 180 to 0 degrees) of 13 muscles.*

5.1.2 Individual Muscles

5.1.2.1 Anterior Deltoid

The peak amplitude for AD occurs at 69% in Phase 1, and at 55% in Phase 2 (Figure 5.3). There is a consistent increase in amplitude until the peak is reached in Phase 1, and the second peak occurs to resist gravity and arrest the downward motion of the upper limb.

Figure 5.4 illustrates that the shape of the activation in of this muscle in the supine position, compared to standing and has a different amplitude character. The peak amplitude occurs earlier in Phase 1 at 57%, compared to 68%, whereas the subtler secondary lesser peak in Phase 2 occurs at the same time.

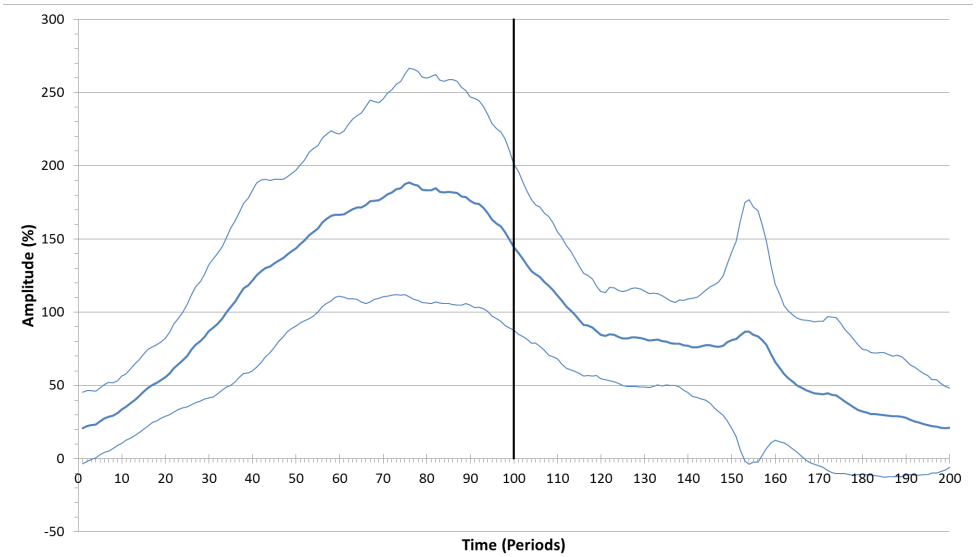


Figure 5.3. Graph to show normal shoulder group (n=19) activation for AD for the movement forward flexion. The thick and thin lines represent the mean amplitude and SD (+/-) respectively. The time period 0-100 represents 0 to 180 degrees in the upstroke and 100-200 represents 180-0 degrees in the down stroke.

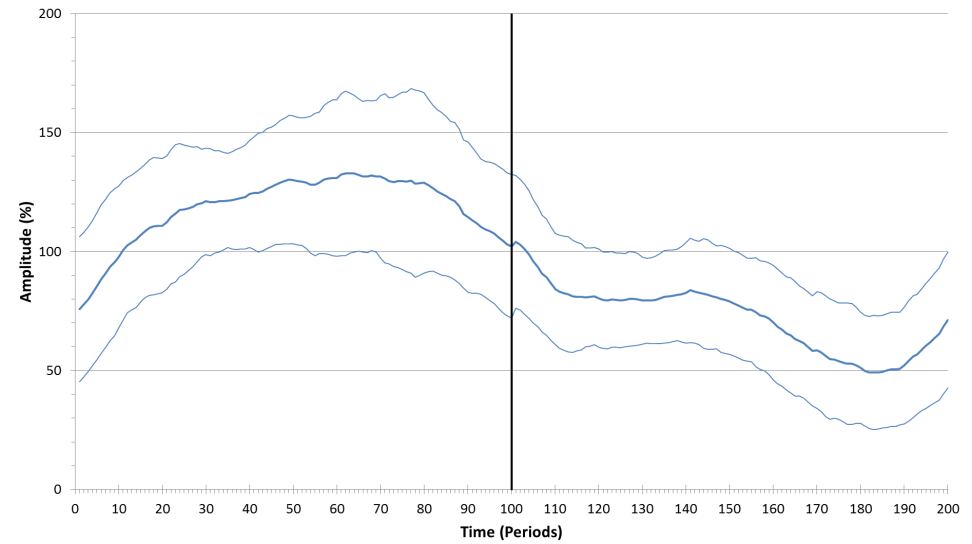


Figure 5.4. Graph to show normal shoulder group (n=22) activation for AD for the movement forward flexion whilst in the supine position. The thick and thin lines represent the mean amplitude and SD (+/-) respectively. The time period 0-100 represents 0 to 30 degrees in the upstroke and 100-200 represents 30-0 degrees in the down stroke.

5.1.2.2 Middle Deltoid

Appendix 1 – Figure A1.3 for the standing forward flexion and Figure A1.4 for the same movement in the supine position.

As with AD, there is a consistent increase in amplitude until the peak is reached at 72% of Phase 1 movement. In Phase 2, there is no secondary peak, the amplitude rapidly decreases in the first 30% of Phase 2 and then the reduction almost reaches a plateau. Of the three deltoid muscles, MD exhibited the highest amplitude.

Compare the amplitude for the two positions of standing and supine. The amplitude pattern is very different with a plateau during each phase rather than a peak and then gradual reduction.

5.1.2.3 Posterior Deltoid

Appendix 1 – Figure A1.5 for the standing forward flexion and Figure A1.6 for the same movement in the supine position.

For the standing position PD is the third part of the deltoid muscle [320, 321], the most posterior portion, thus in Phase 1 the increase in amplitude follows a similar pattern to AD and MD (Figure 5.3 and A1.3). However, the amplitude is the least of the three deltoid muscles, at 110%. The peak amplitude of all three muscles occurs

at around the same point in Phase 1, 68-72%. The secondary peak in Phase 2, which is absent in MD, occurs towards the end of the phase at 80%.

The amplitude pattern in the supine position (Figure A1.6) is shallower with less definitive peaks. The peak in Phase 1 occurs at 58%, which is slightly earlier, compared to the standing position. Comparing the peak amplitude in both Phases, their timing is approximately the same.

5.1.2.4 Upper Trapezium

Appendix 1 – Figure A1.7 for the standing forward flexion and Figure A1.8 for the same movement in the supine position.

In Phase 1, the peak amplitude for UT (Figure A1.7) occurs at 52%, earlier than for the three parts of deltoid. The second peak in Phase 2 occurs around the same time as AD (Figure 5.3) at 53%.

Previously described, Chapter 5.1.3.3, muscles in the supine position (Figure A1.8) show the intensity of activation in a more sustained and constant in character. Although less obvious, the peak of activation occurs at roughly the same point in both phases, around the 40% mark in Phase 1 and 50% in Phase 2.

5.1.2.5 Serratus Anterior

Appendix 1 – Figure A1.09 for the standing forward flexion and Figure A1.10 for the same movement in the supine position.

In standing position, the gradient of Phase 1 is shallower that for other muscles already considered. There is a primary peak towards the end of phase 1; however, there is also a secondary peak at 42%. Phase 2 illustrates a flatter gradient decline, with a slight peak occurring toward the end of the phase at 80%.

The amplitude has a different pattern for the supine position (19), with less amplitude and two subtle peaks occurring at approximately 50% during both phases.

5.1.2.6 Teres Major

Appendix 1 – Figure A1.11/ for the standing forward flexion and Figure A1.12 for the same movement in the supine position.

The recordings of this muscle were problematic in the standing position; this can be particularly seen in Phase 2, where there are three periods of extremely large variation, which is attributable to artifact. Phase 1 shows a peak at 69%, but in light of the large

variation in Phase 2, one cannot be confident of the true pattern, although it seems to show a gradual decrease in amplitude.

Figure 5.5 illustrates the amplitude pattern for TM in the supine position. The amplitude is shallower and the peak in Phase 1 is toward the end of the phase and at 50% during Phase 2.

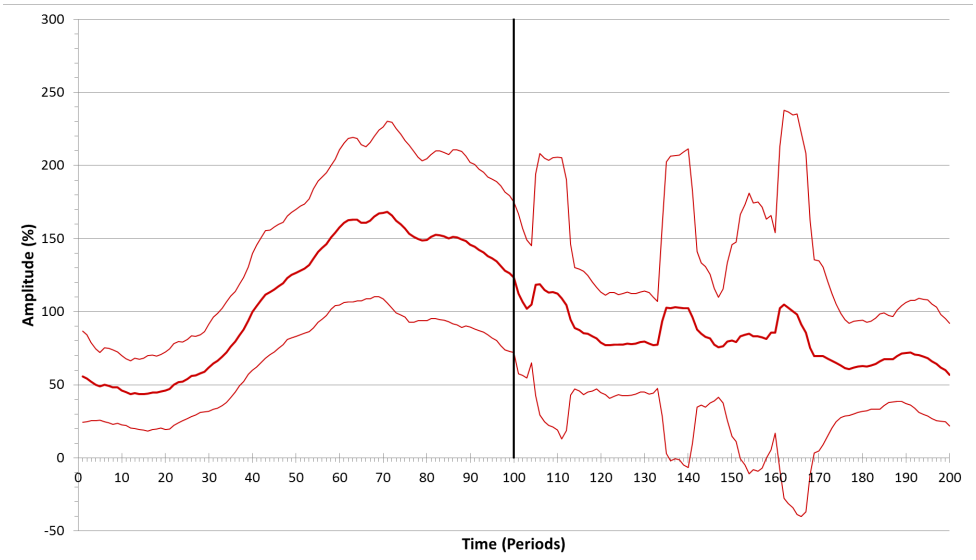


Figure 5.5. Graph to show the normal shoulder group (n=13) activation for TM for the movement forward flexion. The thick line and thin line present the mean amplitude and SD (+/-) respectively. The time period 0-100 represent 0 to 180 degrees in the upstroke and 100-200 represents 180-0 degrees in the down stroke. Note the periods of very high variation in Phase 2 (see text).

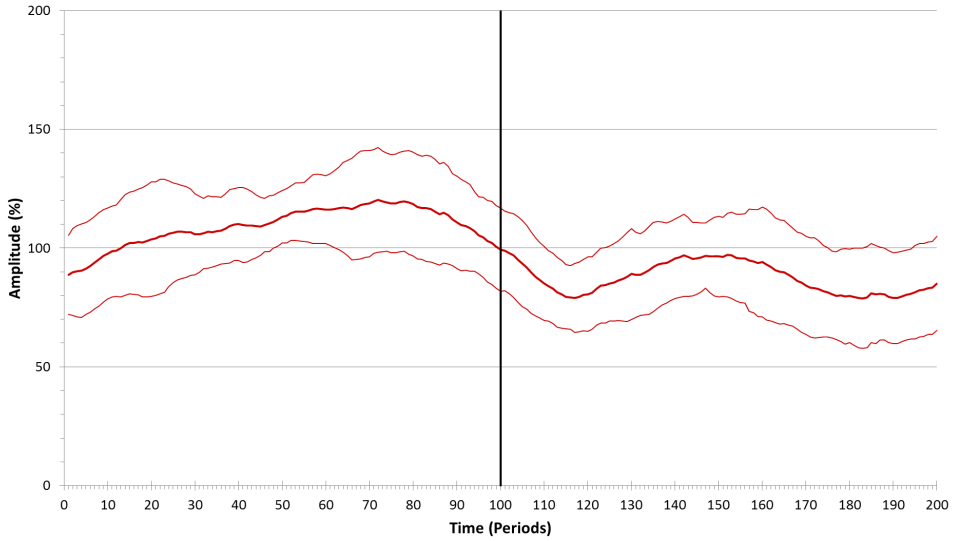


Figure 5.6. Graph to show the normal shoulder group (n=19) activation for TM for the movement forward flexion whilst in the Supine position. The thick line and thin line present the mean amplitude and SD (+/-) respectively. The time period 0-100 represents the upstroke and 100-200 represents the down stroke.

5.1.2.7 Latissimus Dorsi

Appendix 1 – Figure A.13 for the standing forward flexion and Figure A1.14 for the same movement in the supine position.

The pattern of Phase 1 is a gradual increase in amplitude and occurs until the plateau is reached at 40%. At this point a plateau occurs in the region of 110%, which starts to reduce until after 92%. In Phase 2, there is a secondary peak around 57%, followed dip and then slight further rise.

The activation pattern in the supine position is more complicated, (Figure 1.14), however, the amplitude is greater in Phase 1 compared to the other Phase. Further, in Phase 2 the peak amplitude occurs around the midpoint but in both is not pronounced.

5.1.2.8 Pectoralis Major

Appendix 1 – Figure A1.15 for the standing forward flexion and Figure A1.16 for the same movement in the supine position.

As Figure 5.17 demonstrates for PM, the peak amplitude in the standing position is around 37% in Phase 1 and the further peak at around 51%. This pattern is similar to the UT muscle with peak activations occurring at similar times.

The amplitude in the supine position is similar to that of LD with reasonably consistent amplitude throughout the movement in Phase 1, and a peak at 50%.

5.1.2.9 Biceps Brachii

Appendix 1 – Figure A1.19 for the standing forward flexion and Figure A1.20 for the same movement in the supine position.

Figure A1.17 illustrates that in Phase 1, this muscle has a double peak of similar amplitude, at 34% and 91%. Phase 2 is marked by a gradual decrease in amplitude with a shallow peak at around 45%.

In the supine position, there are also three peaks across the two phases, although the maximum peak occurs in the first peak in the supine position, which is comparative to the second in the standing position.

5.1.2.10 Supraspinatus

Appendix 1 – Figure A1.19 for the standing forward flexion and Figure A1.20 for the same movement in the supine position.

It can be seen in Figure A1.19 that there is a sharp rise in amplitude until 30 followed by a more gradual decline. In Phase 2 there is an even rise and fall around a peak of amplitude at around 50%. Interestingly, the peak in Phase 2 occurs at the same time as AD (Figure 5.3).

The pattern of SSP in the supine position is similar, with an initial peak early in Phase 1, although the second peak of amplitude occurs slightly earlier.

5.1.2.11 Infraspinatus

Appendix 1 – Figure A1.21 for the standing forward flexion and Figure A1.22 for the same movement in the supine position.

Figure A1.21 illustrates that up to 40% there is a rapid increase in amplitude that then flattens for most of the rest of Phase 1. There are two peaks within Phase 2, the largest at 55% and the other shallower peak at 32%. Phase 1 is similar in appearance to Phase 1 of LD (Figure A1.13).

In the supine position the pattern of the amplitude is very different (Figure 5.24) for Phase 1, with a peak during the initial part of this phase and then a gradual reduction. There is a peak in Phase 2 at approximately the same time although the character of amplitude is different.

5.1.2.12 Subscapularis

Appendix 1 – Figure A1.23 for the standing forward flexion and Figure A1.24 for the same movement in the supine position.

Figure 5.25 illustrates that similar to other muscles, ISP and LD in the standing position, there is a plateau of amplitude intensity

although not as pronounced. Phase 2 demonstrates gradual decrease intensity.

The character of SSP in the supine position is plateau-like in character, with the amplitude comparatively higher in Phase 1. The amplitude baseline is greater at the start of Phase 1 and end of Phase 2, which is similar for all the muscles previously described except PD.

5.2 Comparative Study – Patient and Controls – Forward Flexion

5.2.1 Mean Activations

Table 5.3 and 5.4 illustrate the overall amplitudes for the 10 muscles for the patient and the control group. The mean amplitude is greater in the patient group compared to the control for both phases. In Phase 1 for the control group the mean is 59% (SD 11, range 34) compared to the patient group 97% (SD 7, range 25). In Phase 2 the mean for the control group 29% (SD 19, range 63) and for the patient group the mean is 90% (SD 7., range 25).

Figure 5.7 and Figure 5.8 illustrate that for the patient group the difference for the means between the phases is not as pronounced

compared to the controls. This is confirmed by the lack of statistical difference between any of the muscles in the patient group between the phases. The controls show a significant difference in the muscles AD, MD, LD, BB and ISP between the two phases.

5.2.1.1 Patient Group

*Table 5.3. Table reporting the mean signal amplitude of the patient group during the movement of forward flexion during phase 1, (upward vertical movement from 0 to 90 degrees); and phase 2, (down movement from 90 to 0 degrees) of thirteen muscles. SEM is the standard error of measurement. *The t-test shown, assessed whether there was a difference between the two phases.*

		Phase 1			Phase 2		
Muscles	n	Mean (%)	SEM	n	Mean (%)	SEM	t test *
AD	14	101.1	9.9	14	84.1	10.0	0.177
MD	14	100.9	9.6	14	85.4	9.9	0.229
PD	14	96.3	6.3	14	103.7	7.1	0.379
UT	14	100.7	7.8	14	89.6	7.7	0.395
SA	14	91.7	9.6	14	78.2	9.1	0.215
TM	14	93.1	9.4	14	92.1	10.6	0.942
LD	14	86.3	8.7	14	88.8	9.4	0.815
PM	14	111.8	13.7	14	100.0	14.3	0.278
BB	14	92.6	9.9	14	91.1	9.9	0.928
ISP	14	90.5	8.1	14	90.7	8.1	0.982

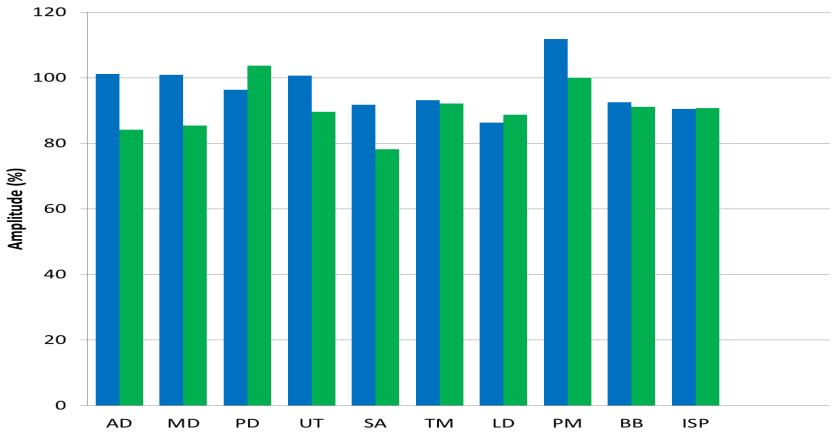


Figure 5.7. Graph to *the mean signal amplitude of the Patient group during the movement of Forward Flexion during phase 1(Blue), (upward vertical movement from 0 to 90 degrees), and phase 2(Green), (down movement from 90 to 0 degrees) of thirteen muscles during the standing forward flexion.*

5.2.1.2 Control Group

*Table 5.4. Table reporting the mean signal amplitude of the Control group during the movement of forward flexion during phase 1, (upward vertical movement from 0 to 90 degrees); and phase 2, (down movement from 90 to 0 degrees) of thirteen muscles. SEM is the standard error of measurement. *The t-test shown, assessed whether there was a difference between the two phases.*

	Phase 1			Phase 2			
Muscles	n	Mean (%)	SEM	n	Mean (%)	SEM	t test *
AD	10	62.4	6.7	10	18.3	9.7	0.016
MD	12	72.8	5.6	12	16.6	8.2	0.001
PD	9	43.2	12.2	9	71.0	11.6	0.222
UT	10	38.4	6.0	10	30.5	8.9	0.558
SA	9	50.8	5.8	9	39.3	9.8	0.471
TM	11	60.6	6.7	11	40.6	12.1	0.209
LD	9	64.2	8.0	9	23.9	10.5	0.042
PM	8	63.6	4.9	8	29.4	12.6	0.074
BB	11	69.0	8.5	11	7.9	3.0	0.000
ISP	10	61.1	4.7	10	16.6	8.0	0.002

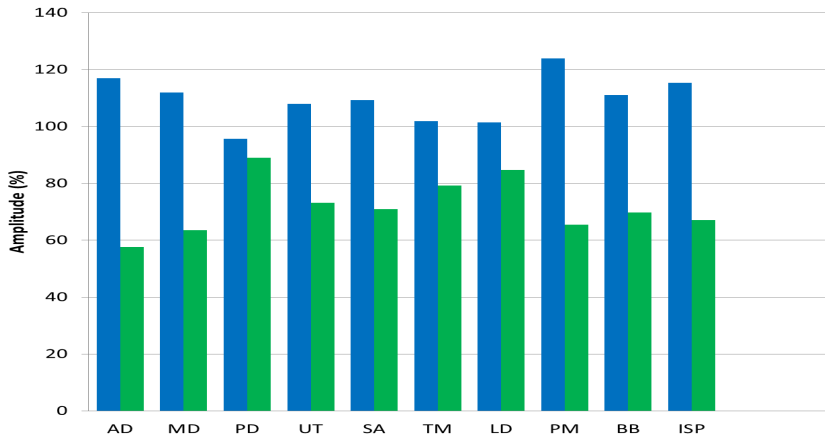


Figure 5.8. Graph to *the mean signal amplitude of the Control group during the movement of forward flexion during phase 1(Blue), (upward vertical movement from 0 to 90 degrees), and phase 2(Green), (down movement from 90 to 0 degrees) of thirteen muscles.*

5.2.2 Comparison of muscle activation patterns in forward flexion

Muscle activation patterns are shown throughout this chapter as amplitude plotted against time. However, the peak amplitude gives an objective single measure that can be used as a marker for the pattern of activation.

Figure 5.9 and 5.10 illustrate the relative timings both in Phase 1 and Phase 2 for both the patient and control group. In Phase 1 AD, MD, BB and ISP the peak amplitude occurs earlier in the patients compared to the controls. By contrast, for PD, UT and SA, the reverse is true with the patients' activations occurring later than the controls'.

In Phase 2 the pattern is dissimilar compared to Phase 1, with patient activations occurring earlier in AD, PD only, and later in UT, SA, TM, BB and ISP. There is a significant difference across the 10 muscles in the time of the peak amplitudes (p=0.012).

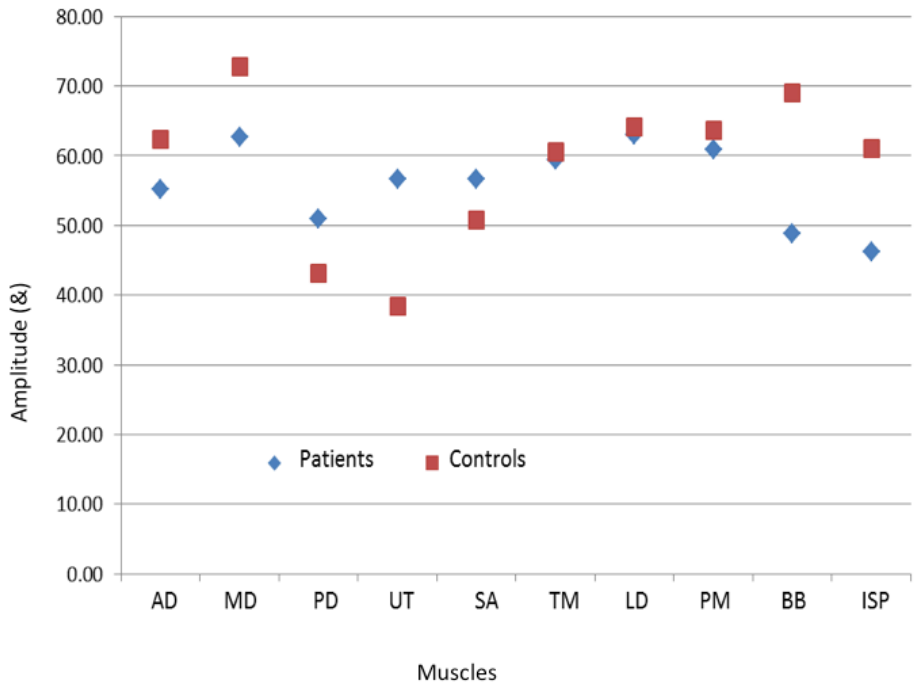


Figure 5.9. Graph to show the time of maximum amplitude within Phase 1 (phase length 0-100) for forward flexion comparing the patient and control group (p=0.26)

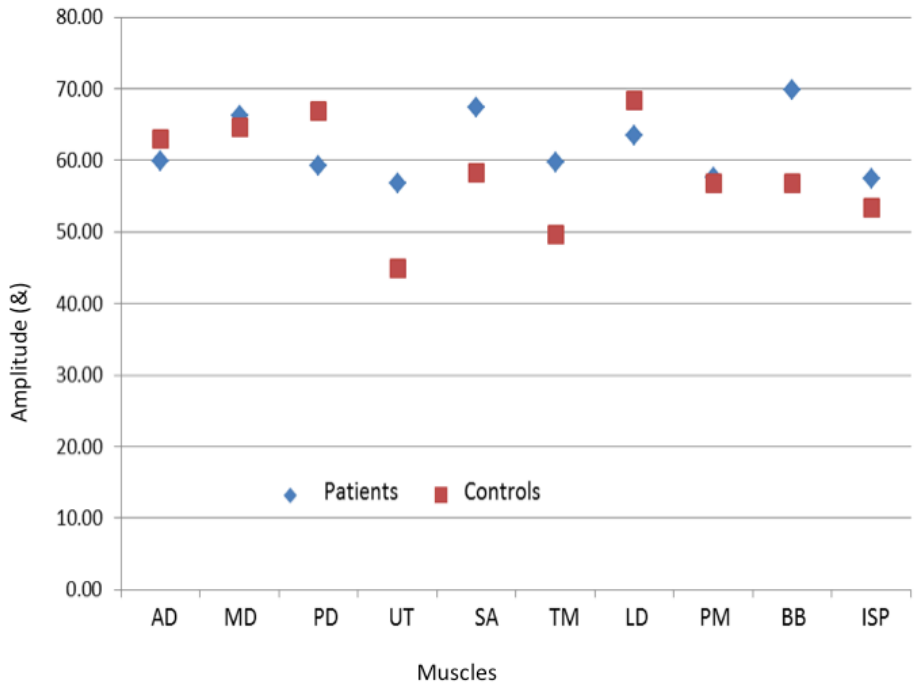


Figure 5.10. Graph to show the time of maximum amplitude within Phase 2 (phase length 0-100) for forward flexion comparing the patient and control group (p=0.012)

5.2.3 Individual Muscles

The amplitude patterns, Figure A2.1 – A2.11, for the individual muscles in this section these are for the movement forward flexion from 0-90 degrees. This is contrast to the amplitudes set out in section 5.1, which are in response to the greater movement 0-180 degrees.

As can be seen for the entire patient group, the amplitude percentages are more variable, as evidenced by there overall larger and less consistent standard deviation. Frequently when there is a peak of amplitude, the peak is biphasic, which will be explored in the next chapter; this will not be described throughout the individual muscle section that follows.

The individual muscles activations for all the muscles tested is set out in Appendix 2, some of these activations is set out for illustrative purposes.

5.2.3.1 Anterior Deltoid

Figure 5.11 illustrates the amplitudes for the patient group, and Figure 32 shows the pattern for the control. There is a stark difference both in terms of pattern and character. The pattern of activation for the control group is similar to that of the normal shoulder group (Figure 5.3) although there is no discernable secondary peak in Phase 2. For the patient group there is a greater consistently higher amplitude, with two peaks, one in each phase.

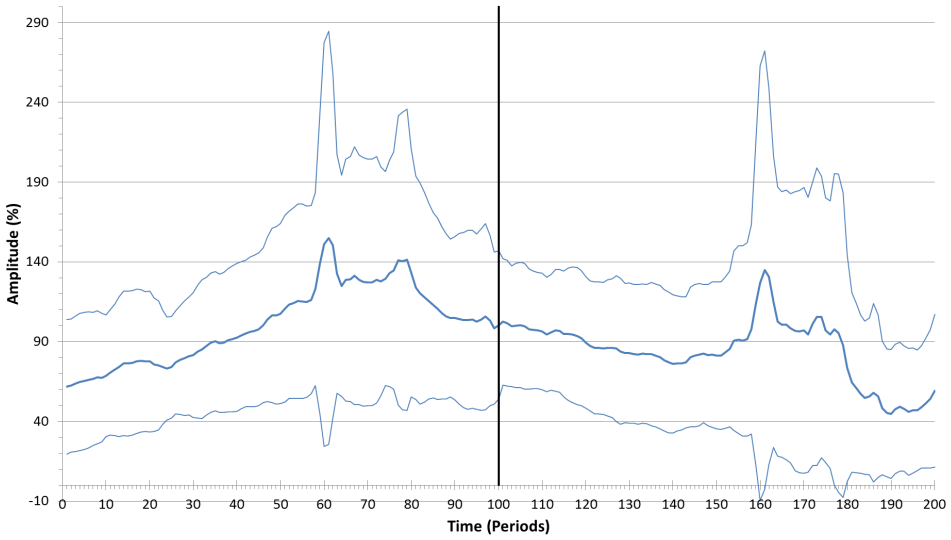


Figure 5.11. Graph to show the Patient group (n=14) activation for AD for the movement forward flexion. The thick line and thin line present the mean amplitude and SD(+/-) respectively. The time period 0-100 represent 0 to 90 degrees in the upstroke and 100-200 represents 90-0 degrees in the down stroke.

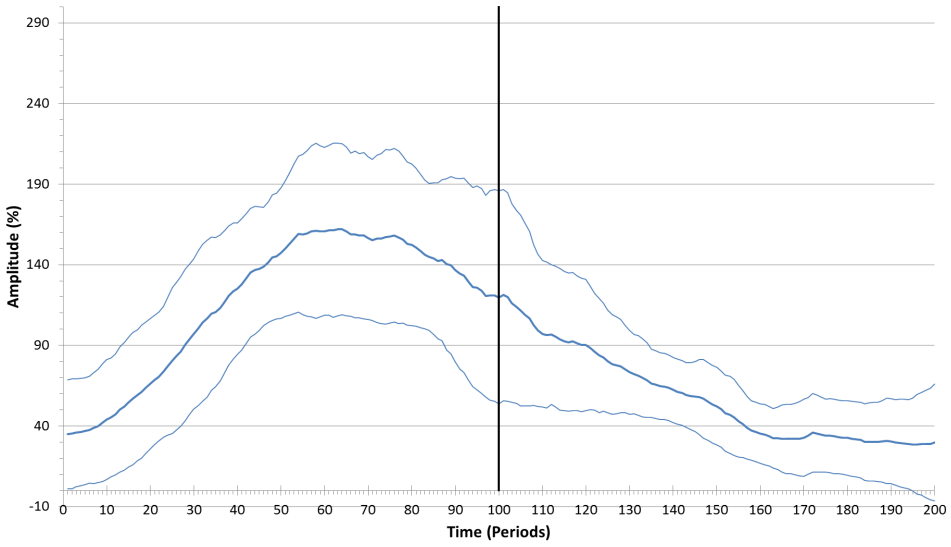


Figure 5.12. Graph to show the Control group (n=10) activation for AD for the movement forward flexion. The thick line and thin line present the mean amplitude and SD (+/-) respectively. The time period 0-100 represent 0 to 90 degrees in the upstroke and 100-200 represent 90-0 degrees in the down stroke.

5.2.3.2 Middle Deltoid

Appendix 2 – Figure A2.3 for the forward flexion of the patient group and Figure A2.4 for the same movement in control group.

The pattern for both groups for MD (Figure 33 and 34) shows a similar contrast to AD.

5.2.3.3 Posterior Deltoid

The pattern for PD for the patient and control group has greater similarity compared to AD and MD. However, the pattern in both Phase 1 and Phase 2 shows, instead of a uniform rise in amplitude, multiple peaks when it increased.

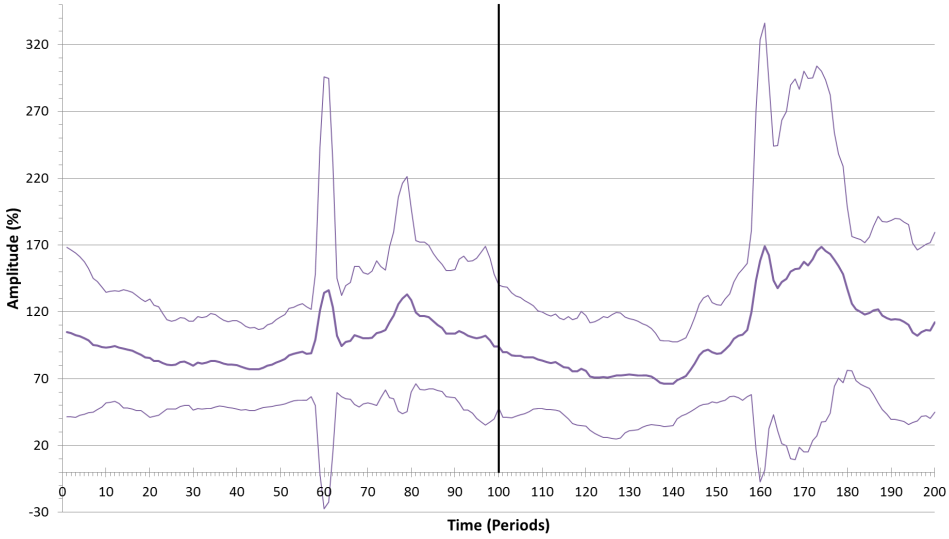


Figure 5.5. Graph to show the Patient group (n=14) activation for PD for the movement forward flexion. The thick line and thin line present the mean amplitude and SD(+/-) respectively. The time period 0-100 represent 0 to 90 degrees in the upstroke and 100-200 represents 90-0 degrees in the down stroke.

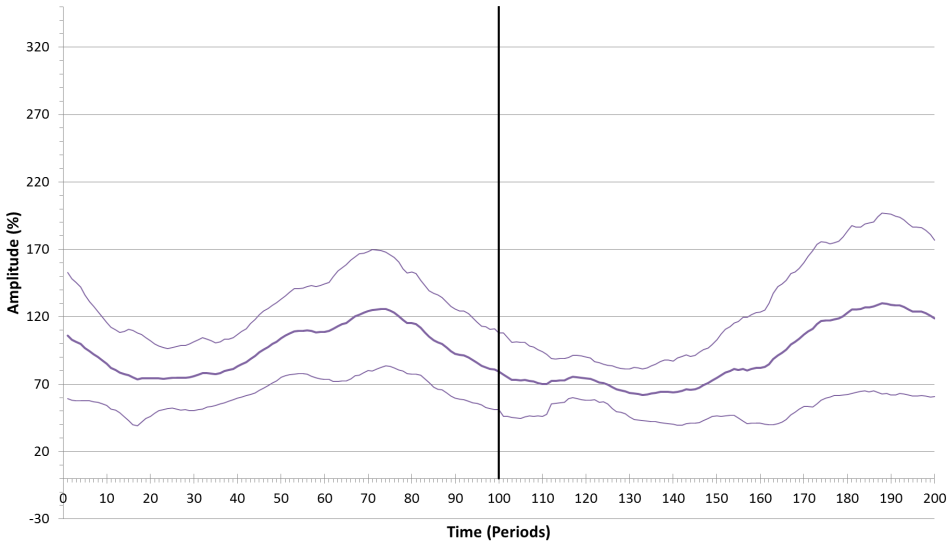


Figure 5.6. Graph to show the Control group (n=9) activation for PD for the movement forward flexion. The thick line and thin line present the mean amplitude and SD (+/-) respectively. The time period 0-100 represent 0 to 90 degrees in the upstroke and 100-200 represent 90-0 degrees in the down stroke.

5.2.3.4 Upper Trapezium

The patient group (Figure 5.15), have an activation pattern that has an on/off character across the two phases. This is in marked contrast to the pattern exhibited by the control group (Figure 5.16), which has a peak earlier on in Phase 1, which is similar to the greater movement of 180 degrees shown in Figure A1.7.

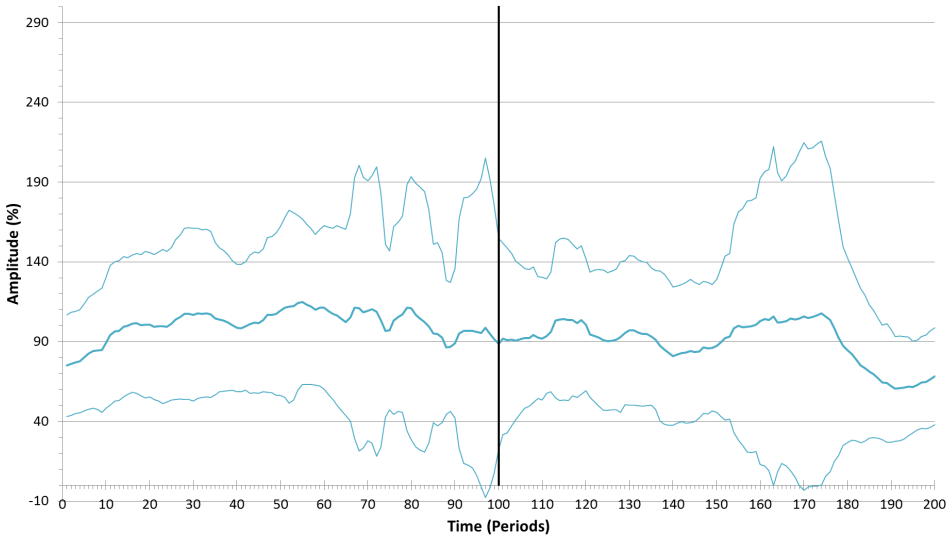


Figure 5.15. Graph to show the Patient group (n=14) activation for UT for the movement forward flexion. The thick line and thin line present the mean amplitude and SD (+/-) respectively. The time period 0-100 represent 0 to 90 degrees in the upstroke and 100-200 represent 90-0 degrees in the down stroke.

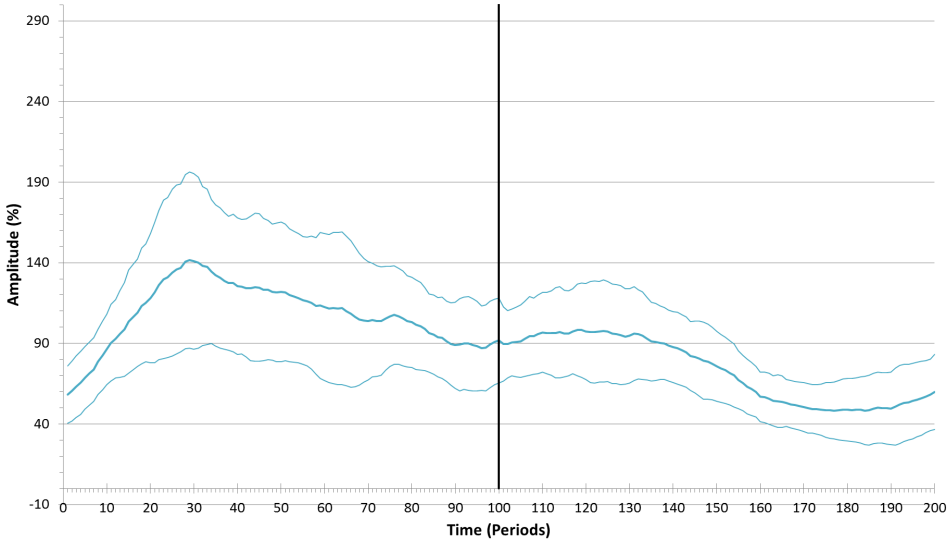


Figure 5.16. Graph to show the Control group (n=10) activation for UT for the movement forward flexion. The thick line and thin line present the mean amplitude and SD (+/-) respectively. The time period 0-100 represent 0 to 90 degrees in the upstroke and 100-200 represents 90-0 degrees in the down stroke.

5.2.3.5 Serratus Anterior

Appendix 2 – Figure 2.9 for the forward flexion of the patient group and Figure 2.10 for the same movement in control group.

As illustrated by Figure A2.9, the amplitude follows a very different pattern for the patient group compared to the controls (Figure A2.10).

5.2.3.6 Teres Major

Appendix 2 – Figure A2.11 for the forward flexion of the patient group and Figure A2.12 for the same movement in control group.

As seen in Figure A2.11, in the patient group, in Phase 2, there is a marked biphasic peak, which is absent in the control group (Figure A2.12). Although the mean amplitude for both groups has a similar baseline, there is greater variation particular in the standard deviation.

5.2.3.7 Latissimus Dorsi

Appendix 2 – Figure A2.13 for the forward flexion of the patient group and Figure A2.14 for the same movement in control group.

Comparing the activation of the patient group, Figure A2.13, to that of the control group, Figure A2.14, there is an additional pronounced peak in Phase 2, which corresponds to a dip in the control group.

Further, variation of the mean amplitude and the standard deviation is particular marked in this muscle.

5.2.3.8 Pectoralis Major

As Figures 5.17 and 5.18 illustrates there is very different activation pattern, in particular during Phase 2. In the control group it can be seen that the amplitude gradually reduces, whereas in the patient group there is a peak around 65%.

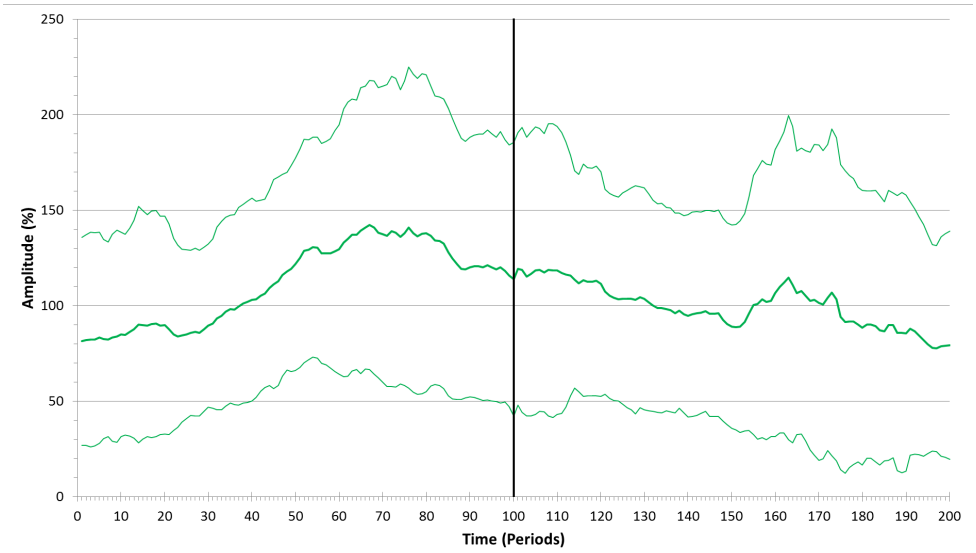


Figure 5.17. Graph to show the Patient group (n=14) activation for PM for the movement forward flexion. The thick line and thin line present the mean amplitude and SD (+/-) respectively. The time period 0-100 represent 0 to 90 degrees in the upstroke and 100-200 represent 90-0 degrees in the down stroke.

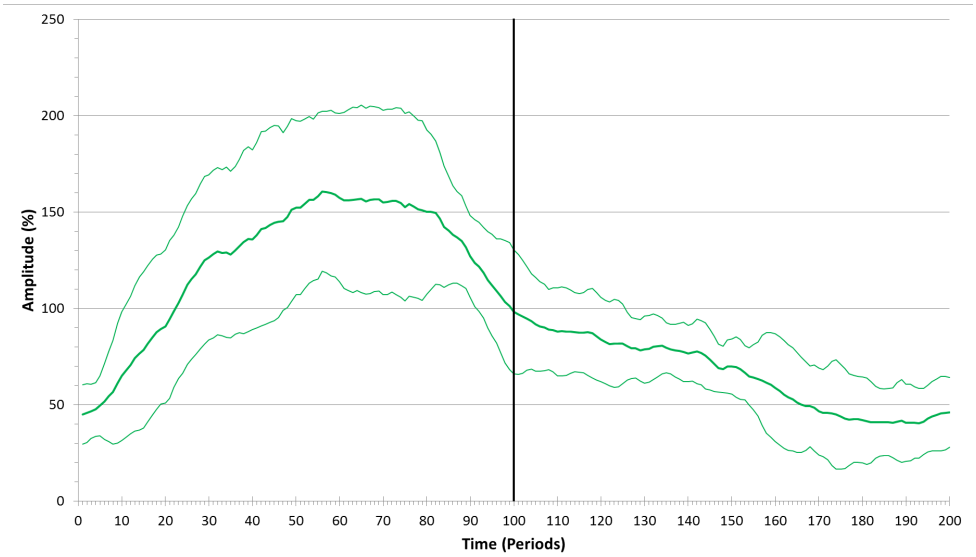


Figure 5.18. Graph to show the Control group (n=8) activation for PM for the movement forward flexion. The thick line and thin line present the mean amplitude and SD (+/-) respectively. The time period 0-100 represent 0 to 90 degrees in the upstroke and 100-200 represent 90-0 degrees in the down stroke.

5.2.3.9 Biceps Brachii

Appendix 2 – Figure A2.17 for the forward flexion of the patient group and Figure A2.18 for the same movement in control group.

For the muscle BB, there is a severe peak in both phases for the patient group (Figure A2.17) compared to the control group (Figure A2.18), which has a gentle peak at around 75% of Phase 1. This additional peak in Phase 2 for the patient group is present in the muscles, PM, LD, TM, SA, MD, ISP and AD.

5.2.3.10 Infraspinatus

Appendix 2 – Figure 2.19 for the forward flexion of the patient group and Figure 2.20 for the same movement in control group.

As previous mentioned in the section above, ISP also has a peak of activation for the patient group (Figure A2.19), which is absent in the control group (Figure A2.20).

It can be seen that there is a marked reduction of activation in the patient group, which shows a plateau, compared to the control group.

5.3 Normal Shoulder Group – Abduction

5.3.1 Global Analysis

5.3.1.1 Standing

Table 5.5 illustrates the mean amplitude for all of the 12 muscles tested, along with a graphical demonstration of the mean amplitude

which is shown in Figure 5.19. In all of the 12 individual muscles there is a significant difference between Phase 1 and Phase 2 (p=<0.001 to p=0.027.

The mean activation between the two phases is pronounced in all but the muscles TM and LD.

The mean amplitude for Phase 1 is 126.83 (SD 9.88, range 29.13), which lowers in Phase 2, where the mean is 78.34 (SD 7.48, range 24.07).

*Table 5.5. Table reporting the mean signal amplitude of the normal shoulder group during the movement of forward flexion whilst standing during phase 1, (upward vertical movement from 0 to 180 degrees); and phase 2, (down movement from 180 to 0 degrees) of thirteen muscles. SEM is the standard error of measurement. *The t-test shown, assessed whether there was a difference between the two phases.*

	Phase 1			Phase 2			
Muscles	n	Mean (%)	SEM	n	Mean (%)	SEM	t test *
AD	19	139.5	3.4	19	70.8	9.0	0.000
MD	18	139.7	3.1	18	69.2	7.2	0.000
PD	18	122.4	2.7	18	81.2	4.3	0.000
UT	17	137.3	4.0	17	74.8	8.5	0.000
SA	16	127.2	3.8	16	78.1	5.7	0.000
TM	14	111.3	3.6	14	91.8	3.8	0.027
LD	11	110.6	2.8	13	89.7	2.8	0.006
PM	11	120.9	2.8	11	81.4	4.1	0.000
BB	12	128.8	5.3	12	75.0	7.1	0.001
SSP	17	125.3	3.2	17	81.2	6.1	0.000
ISP	13	124.1	6.0	13	79.0	6.8	0.006
SUB Scap	8	134.8	3.7	8	67.7	6.1	0.000

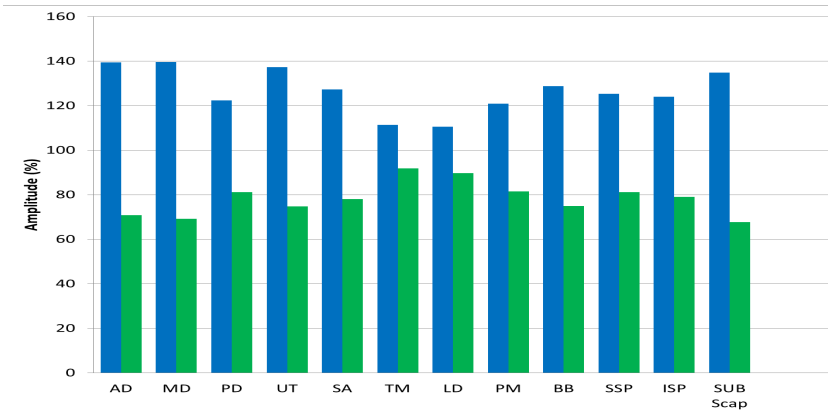


Figure 5.19. Graph to *the mean signal amplitude of the normal shoulder group during the movement of forward flexion whilst standing during phase 1(Blue), (upward vertical movement from 0 to 180 degrees), and phase 2(Green), (down movement from 180 to 0 degrees) of thirteen muscles.*

5.3.1.2 Supine

As stated in the previous section, when considering the following supine data, it is important to appreciate that it is obtained over a small range of motion, approximately 15 degrees.

There is less distinct pattern of mean amplitudes, compared to the supine forward flexion where four muscles (AD, MD, ISP and SUB) had comparatively greater amplitude. In Phase 1 the mean amplitude was 102.09 (SD 5.78, range 20.92), and in Phase 2 the mean was 94.55 (SD 6.47, range 22.95). When compared to the standing abduction data, Phase 1 has greater amplitude and Phase has a lesser amplitude.

*Table 5.6. Table reporting the mean signal amplitude of the normal shoulder group during the movement of abduction/adduction whilst supine during phase 1, (upward vertical movement from 0 to 20 degrees); and phase 2, (down movement from 20 to 0 degrees) of thirteen muscles. SEM is the standard error of measurement. *The t-test shown, assessed whether there was a difference between the two phases.*

		Phase 1			Phase 2		
Muscles	n	Mean (%)	SEM	n	Mean (%)	SEM	t test *
AD	20	104.7	1.5	20	90.0	2.1	0.000
MD	18	107.9	2.1	18	89.1	2.6	0.000
PD	17	100.8	1.8	17	94.3	1.7	0.064
UT	12	104.1	1.7	12	91.8	2.0	0.004
SA	18	103.6	1.5	18	93.7	2.0	0.003
TM	19	102.3	1.2	19	95.2	1.6	0.005
LD	16	96.6	1.6	16	100.7	1.4	0.179
PM	20	100.3	1.2	20	96.4	1.5	0.130
BB	16	88.2	2.2	16	110.6	2.5	0.000
SSP	11	109.1	1.7	11	87.6	1.2	0.000
ISP	17	105.4	2.4	17	90.8	2.6	0.007
SUB Scap	11	100.5	2.7	11	95.2	2.9	0.325

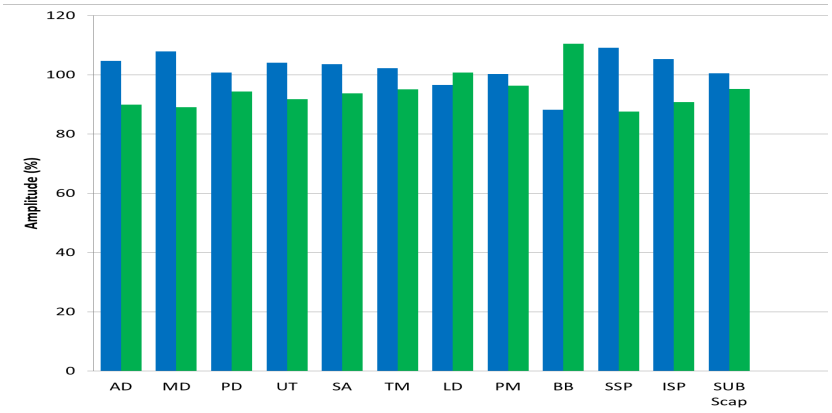


Figure 5.20. Graph to *the mean signal amplitude of the normal shoulder group during the movement of abduction/adduction whilst supine during phase 1(Blue), (upward vertical movement from 0 to 20 degrees), and phase 2(Green), (down movement from 20 to 0 degrees) of thirteen muscles.*

5.3.2 Individual Muscles

5.3.2.1 Anterior Deltoid

Figure 5.21 illustrates the AD muscle activation in the standing position. It can be seen there is a large peak of a biphasic quality centered on 60% of Phase 1, and a secondary peak around the middle of Phase 2.

It can be seen in Figure 5.22 that in the supine position the activation comparatively reduced and more horizontal in profile. However, as with supine forward flexion (Figure 5.4) there is subtle peak in Phase 2.

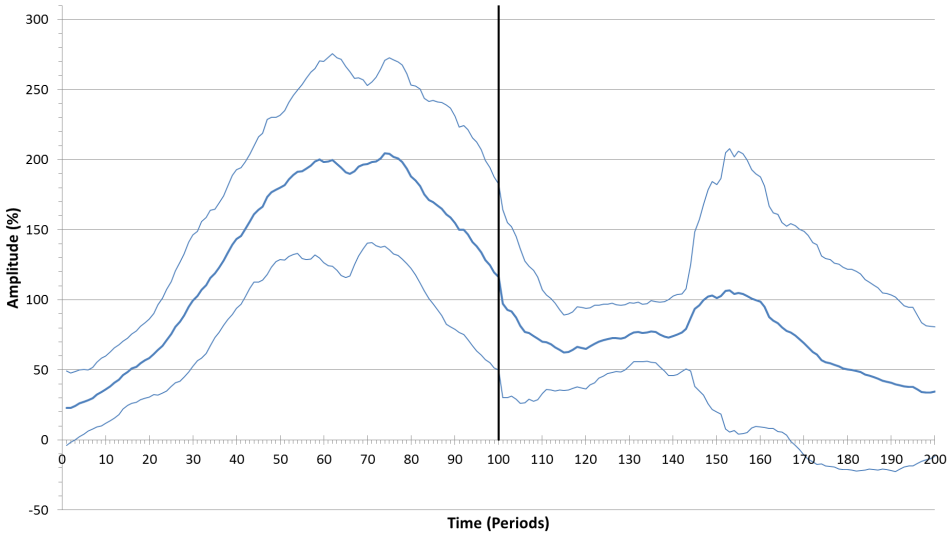


Figure 5.21. Graph to show the normal shoulder group (n=19) activation for AD for the movement abduction. The thick line and thin line present the mean amplitude and SD (+/-) respectively. The time period 0-100 represent 0 to 180 degrees in the upstroke and the time period 100-200 represent 180-0 degrees in the down stroke.

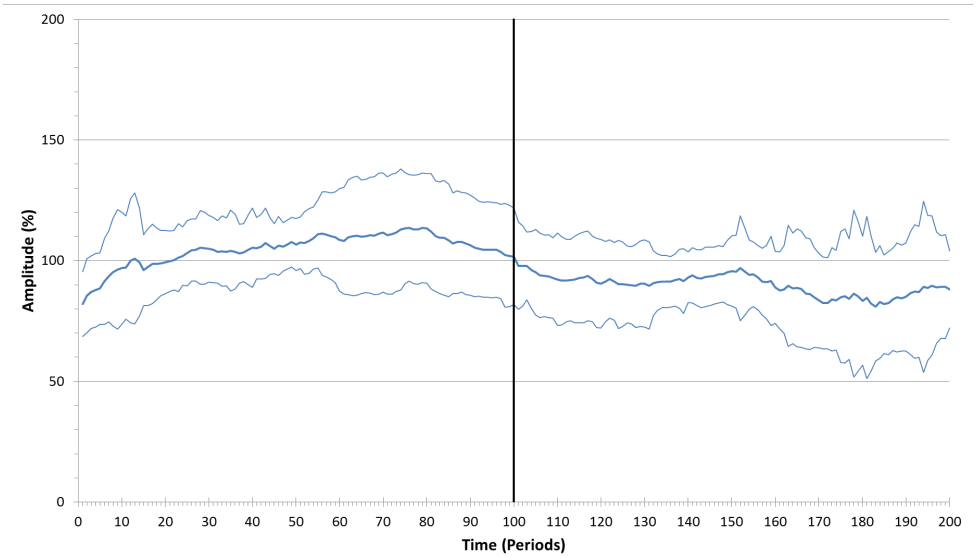


Figure 5.22. Graph to show the normal shoulder group (n=20) activation for AD for the movement abduction/adduction whilst in the Supine position. The thick line and thin line present the mean amplitude and SD (+/-) respectively. The time period 0-100 represents the upstroke and the time period 100-200 represents the down stroke.

5.3.2.2 Middle Deltoid

Appendix 3 – Figure A3.3 for the standing abduction/adduction and Figure A3.4 for the same movement in the supine position.

Figure A3.3, illustrates that in the standing position that there is a consistent increase in amplitude until the peak at 78%, then a gradual decline during Phase 2.

In the supine position the activation is shallower, with less amplitude. There is a small peak in Phase 2.

5.3.2.3 Posterior Deltoid

Appendix 3 – Figure A3.5 for the standing abduction/adduction and Figure A3.6 for the same movement in the supine position.

The activation pattern of PD in the standing position, Figure A3.5, is similar to that of the AD, in there is a large peak in Phase 1 and a second smaller but substantial peak in Phase 2.

Illustrated in Figure A3.6 is the activation for PD in the supine position, as would be expected, there is an increase in Phase 1, which is less pronounced. Note that the 15 degrees of movement for both phases is divided by 100, whereas for the standing range of movement 180 degrees is divided; obviously this will affect the appearance of the graph irrespective of the data recorded.

5.3.2.4 Upper Trapezium

Appendix 3 – Figure A3.7 for the standing abduction/adduction and Figure A3.8 for the same movement in the supine position.

In Phase 1, the peak amplitude for UT (Figure A3.7) occurs at 40.8 which is the earliest point of all 13 muscles tested. There is a secondary small peak in Phase 2 at a similar point.

The supine amplitude shows activation throughout both phases, with higher activation in the Phase 1.

5.3.2.5 Serratus Anterior

Appendix 3 – Figure A3.9 for the standing abduction/adduction and Figure A3.10 for the same movement in the supine position.

Figure A3.9 demonstrates the amplitude for SA in the standing position for abduction. There are two peaks in Phase 1 and one in Phase 2. This is almost a mirror-image of the activations of this muscle in forward flexion, save that the peak in Phase 2 is less pronounced.

The amplitude in the supine position, Figure A3.10, is similar to that of the previous muscles described.

5.3.2.6 Teres Major

Appendix 3 – Figure A3.11 for the standing abduction/adduction and Figure A3.12 for the same movement in the supine position.

In the standing position measuring the movement of abduction to 180 degrees, it can be readily seen if Figure A3.11 is compared to forward flexion in the supine position shown in Figure A1.3.

In the standing position, there is a peak around 72% in Phase 1 and there is a double peak toward the end of Phase 2. In the supine position there is an similar picture to the previous muscle described, namely a small shallow peak in Phase 1, with less amplitude in Phase 2, with a small shallow peak around 65% throughout the phase.

5.3.2.7 Latissimus Dorsi

Appendix 3 – Figure A3.13 for the standing abduction/adduction and Figure A3.14 for the same movement in the supine position.

The muscle amplitude in abduction, Figure A3.13, is considerably different to forward flexion, Figure A1.13.

There is a gradual increase in amplitude with peaks at 76.5% along Phase 1, with a secondary peak at the 90% mark of Phase 2. In the supine position there is constant activation around the amplitude of 100%.

5.3.2.8 Pectoralis Major

Appendix 3 – Figure A3.15 for the standing abduction/adduction and Figure A3.16 for the same movement in the supine position.

As Figure A3.15 illustrates the peak amplitude occurs around the 74.9% in Phase 1, with no real discernable peak in Phase 2 which has a plateau like amplitude.

From the supine amplitude graph, Figure A3.16, there is a gradual buildup of amplitude and peak in Phase 1, with a subtle peak in Phase 2, but as with the other supine amplitude pattern, these are shallow.

5.3.2.9 Biceps Brachii

Appendix 3 – Figure A3.17 for the standing abduction/adduction and Figure A3.18 for the same movement in the supine position.

As shown in Figure A3.17, the amplitude for BB is a single peak in Phase 1 at 78% of that phase and a gradual decline in Phase 2.

The supine data, Figure A3.18, shows a distinctly different pattern compared to the other muscles described in this position with

abduction. There is a large peak in Phase 2 and with the amplitude in Phase 1 it is smaller.

5.3.2.10 Supraspinatus

Appendix 3 – Figure A3.19 for the standing abduction/adduction and Figure A3.20 for the same movement in the supine position.

The pattern illustrated in Figure A3.19 for SSP is almost identical to that for this muscle in the standing position undertaking the movement of forward flexion, Figure A1.19.

In the supine position the movement of abduction has a shallow peak in Phase 1, with a more subtle peak at approximately 50% along the Phase 2 time line.

5.3.2.11 Infraspinatus

Appendix 3 – Figure A3.21 for the standing abduction/adduction and Figure A3.22 for the same movement in the supine position.

The amplitude for ISP is illustrated in Figure A3.21; the pattern is different to that produced by forward flexion in the standing position (Figure 5.23). For abduction there is a peak in Phase 1, with a

shallower peak in Phase 2. For forward flexion there is more of a plateau in Phase 1 and a double peak in Phase 2.

For the supine amplitude the pattern is complicated, the data being erratic. Perhaps the only safe observation is that the amplitude in Phase 1 is higher than in Phase 2.

5.3.2.12 Subscapularis

Appendix 3 – Figure A3.23 for the standing abduction/adduction and Figure A3.24 for the same movement in the supine position.

As illustrated in Figure A3.23, there is a peak in Phase 1 and a gradual decline in Phase 2. This is almost identical to the activations for the movement of forward flexion whilst standing, save that there is a slight peak in the downward trend at the end of Phase 2.

The supine amplitudes demonstrate activations of an erratic nature around the base line of 100% throughout both phases.

5.4 Comparative Study – Patient and Controls – Abduction

5.4.1 Mean Activations

5.4.1.1 Patient Group

The amplitudes for the 10 muscles tested for both the patient and the control group are set out in Table 5.7 and 5.8. Further, the mean amplitudes for these muscles for the two groups are graphically represented in Figure 5.89 and 5.23.

In Phase 1 for the patient group, the mean is 115% (SD 8, range 28) compared to the control group in the same phase of a mean of 118% (SD 12, range 40). Where in Phase 2, the patient group had a mean of 87% (SD 8, range 26), this compares to the control group who had a mean of 80% (SD 14, range 45).

It can be seen that there was a apparent difference for the majority of muscles tested between the two phases. However there was no statistical difference for the muscles of SA, TM and LD for the patient group, compared to TM and LD for the control group.

*Table 5.7. Table reporting the mean signal amplitude of the patient group during the movement of abduction/adduction during phase 1, (upward vertical movement from 0 to 90 degrees); and phase 2, (down movement from 90 to 0 degrees) of thirteen muscles. SEM is the standard error of measurement. *The t-test shown, assessed whether there was a difference between the two phases.*

		Phase 1			Phase 2		
Muscles	n	Mean (%)	SEM	n	Mean (%)	SEM	t test *
AD	12	125.3	6.7	12	79.2	9.1	0.009
MD	12	128.5	5.4	12	74.0	8.0	0.001
PD	12	118.0	2.9	12	86.6	4.8	0.000
UT	12	115.9	4.5	12	86.3	5.6	0.010
SA	11	120.5	9.6	11	81.6	10.4	0.075
TM	11	110.8	6.8	11	88.3	7.5	0.147
LD	12	100.6	3.4	12	99.6	3.8	0.886
PM	12	108.5	3.6	12	96.8	4.5	0.033
BB	12	116.7	6.3	12	86.3	7.9	0.044
ISP	12	115.0	3.0	12	87.8	4.7	0.001

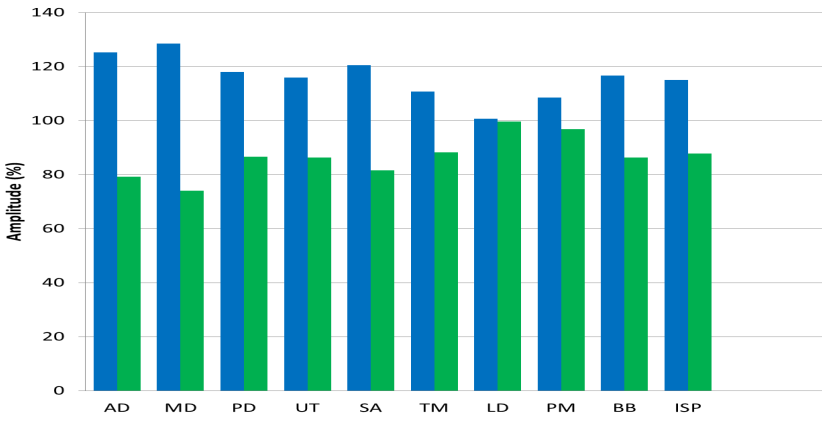


Figure 78. Graph to *the mean signal amplitude of the Patient group during the movement of abduction/abbduction during phase 1(Blue), (upward vertical movement from 0 to 90 degrees), and phase 2(Green), (down movement from 90 to 0 degrees) of thirteen muscles.*

5.4.1.2 Control Group

*Table 5.8. Table reporting the mean signal amplitude of the Control group during the movement of abduction/adduction during phase 1, (upward vertical movement from 0 to 90 degrees); and phase 2, (down movement from 90 to 0 degrees) of thirteen muscles. SEM is the standard error of measurement. *The t-test shown, assessed whether there was a difference between the two phases.*

	Phase 1			Phase 2			
Muscles	n	Mean (%)	SEM	n	Mean (%)	SEM	t test *
AD	12	136.8	2.9	12	58.3	4.7	0.000
MD	13	129.6	5.7	13	65.4	6.7	0.000
PD	12	113.9	4.0	12	85.3	4.8	0.008
UT	13	125.7	5.8	13	73.9	4.8	0.000
SA	10	117.3	4.9	10	80.8	6.7	0.012
TM	8	104.1	6.0	8	94.2	7.5	0.488
LD	9	96.8	5.3	9	103.3	6.7	0.601
PM	9	108.5	2.0	9	90.6	2.2	0.003
BB	10	123.3	5.0	10	72.0	6.0	0.001
ISP	11	120.2	2.4	11	76.2	3.5	0.000

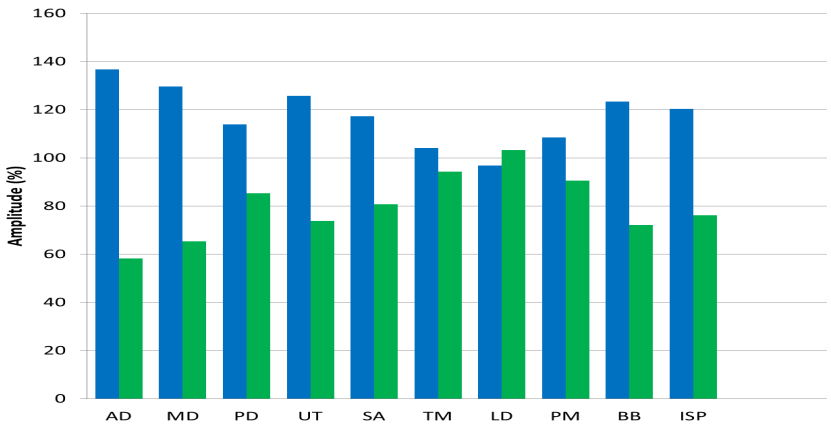


Figure 5.23. Graph to *the mean signal amplitude of the Patient group during the movement of abduction/abbduction during phase 1(Blue), (upward vertical movement from 0 to 90 degrees), and phase 2(Green), (down movement from 90 to 0 degrees) of thirteen muscles.*

5.4.2 Comparison of muscle activation patterns in abduction

Figures 5.24 and 5.25 illustrate the differences between the timing of peak amplitudes for both phases comparing patients versus controls. Overall, there is a statistical difference in time of peak amplitudes, for both Phase 1 ($p=0.04$) and Phase 2 ($p=0.03$).

In Phase 1, for 7 of the muscles, AD, MD, UT, TM, LD, BB and ISP the patient activation is delayed compared to the control group. Further, for the PD, SA and PM the patients' peak activation occurs before the control group.

In Phase 2, for 8 of the muscles, AD, MD, PD, UT, SA, LD, BB and ISP the activation of the patient group, as with the majority in Phase 1, is delayed. With no real difference in the TM peak activation, and the reverse for PM, where the peak activation of the patients occurs before the controls.

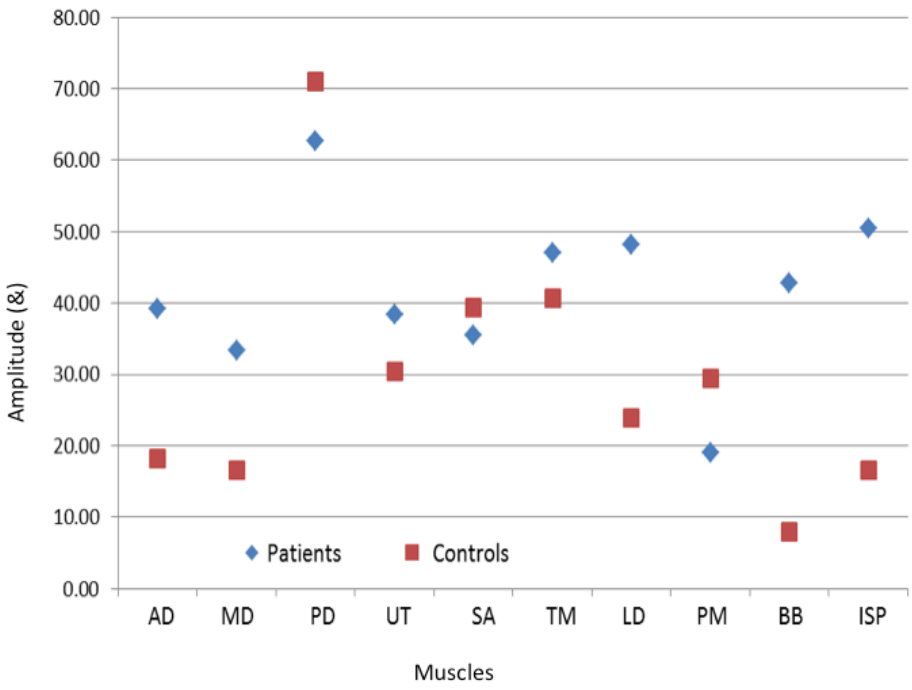


Figure 5.24. Graph to show the time of maximum amplitude within Phase 1 (phase length 0-100) for abduction comparing the patient and control group (p=0.04)

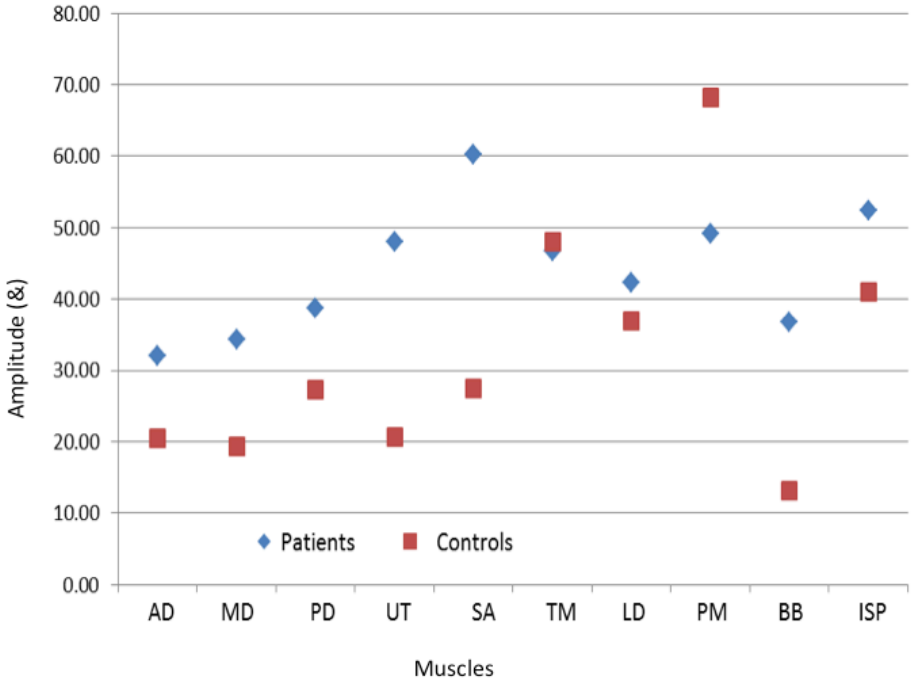


Figure 5.25. Graph to show the time of maximum amplitude within Phase 2 (phase length 0-100) for abduction comparing the patient and control group (p=0.03)

5.4.3 Comparison of individual muscles - Abduction

5.4.3.1 Anterior Deltoid

The amplitude for AD in the patient group is shown in Figure 5.26 and for the control group is shown in Figure 5.27. In Phase 1, it can be observed that the the peak is of lower amplitude and varies in smoothness. In the patient group there is a secondary peak towards the end of Phase 2 that is absent in the control group. The patient group in Phase 2 show such greater changes in gradient, a dramatic change from a downward slope of approximately 30 degrees to to 45 degrees in an upward orientation.

Interestingly in the forward flexion movement, AD, also had a peak in Phase 2 that was absent in the control group.

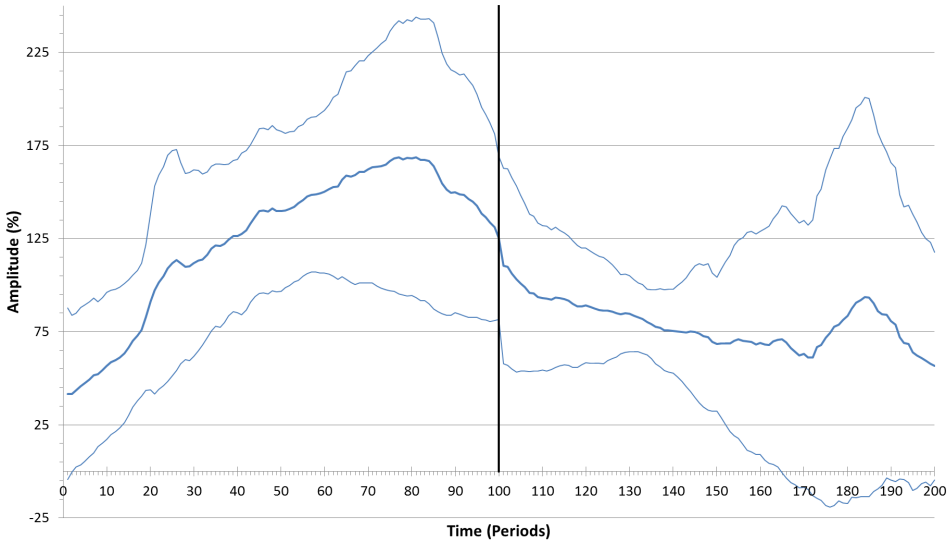


Figure 5.26. Graph to show the Patient group (n=12) activation for AD for the movement adduction/abduction. The thick line and thin line present the mean amplitude and SD (+/-) respectively. The time period 0-100 represent 0 to 90 degrees in the upstroke and 100-200 represent 90-0 degrees in the down stroke.

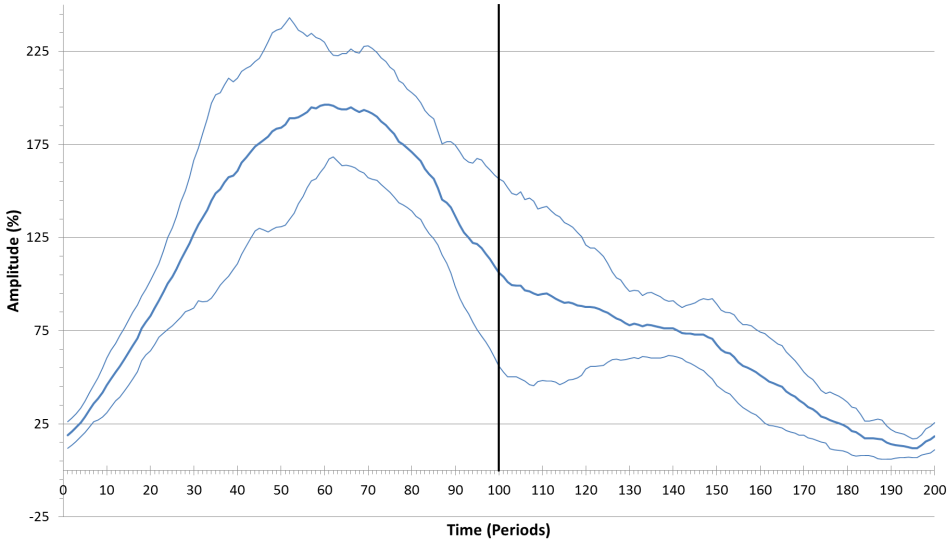


Figure 5.7. Graph to show the Control group (n=12) activation for AD for the movement adduction/abduction. The thick line and thin line present the mean amplitude and SD (+/-) respectively. The time period 0-100 represent 0 to 90 degrees in the upstroke and 100-200 represents 90-0 degrees in the down stroke.

5.4.3.2 Middle Deltoid

Appendix 4 – Figure A4.3 for the abduction/adduction of the patient group and Figure A4.4 for the same movement in control group.

Figure A4.3 illustrates the amplitude of MD in the patient group, and Figure A4.4 shows the amplitude for the control group. There is no major difference between the two groups, but there are subtle differences worthy of comment. The peak in Phase 1 in the patient group is of a different character, namely biphasic, of slightly less amplitude and different gradient.

5.4.3.3 Posterior Deltoid

Appendix 4 – Figure A4.5 for the abduction/adduction of the patient group and Figure A4.6 for the same movement in control group.

The activations for the patient group are shown in Figure A4.5, and for the control group is illustrated in Figure A4.6. The patient group in Phase 1 there is a leveling of the amplitude, which is then maintained for 30% of the phase. This compares to the smooth peak that in the control group.

Further, in Phase 2, there are two more distinct peaks in the patient group compared to a shallow rise in amplitude in the control group. For the patient group the pattern is less erratic in this muscle in this movement compared to the amplitude pattern generate in this group in forward flexion (Figure 5.13).

5.4.3.4 Upper Trapezium

Appendix 4 – Figure A4.7 for the abduction/adduction of the patient group and Figure A4.8 for the same movement in control group.

Figure A4.7 illustrates the difference in Phase 1 activation for the patient group, compared to the same phase for the control group,

Figure A4.8. There is an extended plateau during Phase 1 for the patient group at approximately 125%, whereas for the control group there is a smooth peak, with a maximum of 158%.

The character of Phase 2 is different between the two groups, the gradients are different and the muscle amplitude is maintained at a higher level for the patient group.

5.4.3.5 Serratus Anterior

Figure A1.22 and A2.9 illustrates the amplitude for the patient and the control group respectively. It can be seen that in Phase 1 and 2 that the character of amplitude is rather different. In Phase 1, there is greater activation in the patient group, and the peak amplitude occurs later in the phase. In Phase 2 there are two peaks of amplitude for the patient group compared to a single peak in the control group.

Comparing the activation in SA in forward flexion, the patient group also had two peaks which were both absent in the control group.

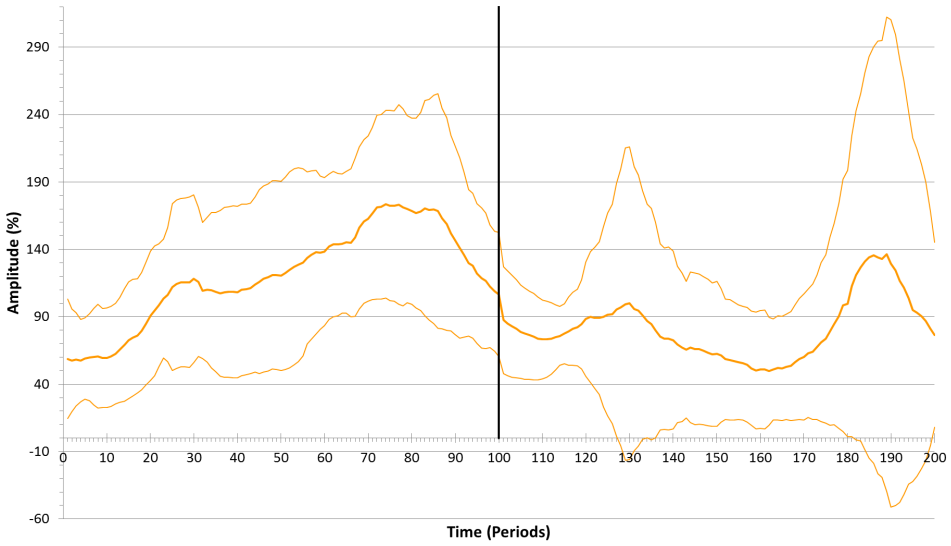


Figure 5.8. Graph to show the Patient group (n=11) activation for SA for the movement adduction/abduction. The thick line and thin line present the mean amplitude and SD (+/-) respectively. The time period 0-100 represent 0 to 90 degrees in the upstroke and 100-200 represents 90-0 degrees in the down stroke.

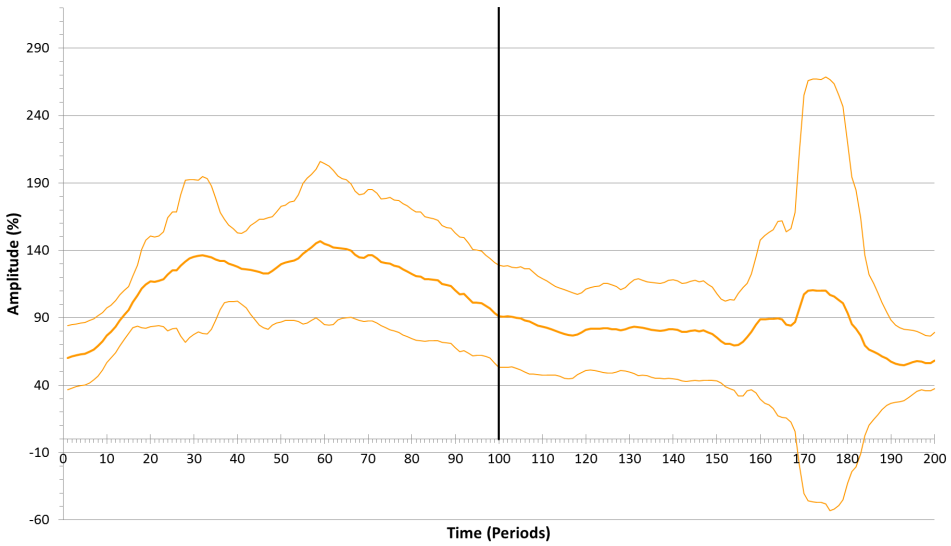


Figure 5.9. Graph to show the Control group (n=10) activation for SA for the movement adduction/abduction. The thick line and thin line present the mean amplitude and SD (+/-) respectively. The time period 0-100 represent 0 to 90 degrees in the upstroke and 100-200 represents 90-0 degrees in the down stroke.

5.4.3.6 Teres Major

Appendix 4 – Figure A4.11 for the abduction/adduction of the patient group and Figure A4.12 for the same movement in control group.

The activations for TM for both patient group (Figure A4.11) and the control group (Figure A4.12) show globally a different pattern. The patient group in Phase 1 has two peaks as opposed to the control group who only has a smoothed single peak. In Phase 2 the patient group has a later peak of increased amplitude comparative to the control group.

5.4.3.7 Latissimus Dorsi

Appendix 4 – Figure A4.13 for the abduction/adduction of the patient group and Figure A4.14 for the same movement in control group.

Figure A4.13 illustrates the amplitude for LD for the patient group, with the amplitude for the control group shown in Figure 4.14. The pattern of activation for both groups is complex however the varying amplitudes are similar. In Phase 1 the patient group's maximum amplitude occurs at approximately the same time, whereas in Phase 2 the control group reaches the maximum earlier in the phase.

5.4.3.8 Pectoralis Major

Appendix 4 – Figure A4.15 for the abduction/adduction of the patient group and Figure A4.16 for the same movement in control group.

For the standing movement of abduction, the activation patterns for the patient group are shown in Figure 5.107 and the control group in Figure A4.16. In Phase 1, the control group has two fairly discernable peaks, whereas the patient group shows an erratic build up to a single peak. In Phase 2 the control group has two peaks, compared to a shallow single peak for the patient group.

5.4.3.9 Biceps Brachii

Appendix 4 – Figure A4.17 for the abduction/adduction of the patient group and Figure A4.18 for the same movement in control group.

As illustrated in Figures A4.17 and A4.18, the amplitude pattern for the movement of abduction produces differences between the groups, but they are not as profound as for the other muscles tested. In Phase 1 the patient group's amplitude is comparatively less and the peak activation occurs later. The most notable difference between the groups in Phase 2, is the substantial peak at the end this phase for the patient group which is absent in the control group.

5.4.3.10 Infraspinatus

Appendix 4 – Figure A4.19 for the abduction/adduction of the patient group and Figure A4.20 for the same movement in control group.

Figure A4.19 illustrates the activation of ISP for the patient group and Figure A4.20 for the control group. For the patient group in Phase 1 the maximum amplitude is achieved later and the build up to that maximum more erratic. In Phase 2 for the patient group there is peak toward the end of the phase that is absent in the control group.

Comparing this movement to that of forward flexion, there is a similar peak in Phase 2, that is absent in the control group.

6 Discussion

There is a large body of data to discuss from the interrelated EMG and fMRI studies. I will focus on the findings that are clinically or physiologically noteworthy, rather than attempting to explain all the results. Later in this chapter I will address the fMRI results, and then the EMG results. Before I do, however, I will discuss the development of the fMRI movement protocol.

6.1 Development of fMRI protocol to assess shoulder movement

6.1.1 Control Group Selection

The control groups were screened for any history of shoulder pathology and the instability questionnaires WOSI and OIS were used as pseudo-objective markers. As illustrated by the scores, Figure 4.1 and 4.2, the control group exhibited no signs of shoulder instability.

A decision was made not to clinically examine the controls/patients prior to the fMRI testing, in order to adopt a standardised approach to the protocol. Clinical experience suggests that even examination of the patients might impede their ability to perform and/or complete

the fMRI or EMG protocols. There was close observation of shoulder movement of the normal group/control group particularly through the EMG protocol that would have exposed any sign of difficulty in shoulder movement, enabling their inclusion to be reviewed. None of the normal controls exhibited any difficulty in undertaking either of the protocols; however 4 of the patients were unable to complete the EMG protocol. The fMRI examination was always undertaken before the EMG protocol.

As addressed in Chapter 2.2.3, the role of lateralisation or "handedness" has received a great deal of attention in research, and is complex. Some work has suggested that lateralisation is an influential factor in the motor cortex on upper limb movement [180, 182, 185, 186], with increased bilateral activation. However, in a recent study of 284 individuals handedness was found not to be influential in motor activations [183]. Spraker et al. has suggested that the asymmetrical response in the motor cortex is a culmination of the ipsilateral innervations contrasted against the transcallosal inhibitory control, and not related to laterality [322]. Further, Hayashi et al. found that there was no significant difference in handedness [184]. This work established that laterality was only a significant issue for repetitive movement and when fine movements of the hand were executed [184]. For the purposes of the present study, therefore, the laterality of both patients and the controls' can be safely disregarded.

In the patient groups it was the unstable shoulder that was moved during the fMRI protocol (Table 3.11); there were 3 left shoulders affected, with the remaining 13 being on the right. In order to avoid limiting the sample size, this variation was eliminated in pre-processing. In SPM a symmetrical version of the TPM was created, by creating an average of the flipped and unflipped TPM, which was then used in the normalisation and segmentation, as set out in greater detail in Chapter 3.4.3. The left-affected patients and controls, whose left shoulder was examined, were flipped prior to the pre-processing.

Age has been identified as a factor that can influence cortical representation [177]. Care was taken to age-match the patients with the controls at least in terms of the mean age (Table 3.11).

6.1.2 Discussion of the practicalities of the protocol

The protocol (Chapter 3.4.2) worked well, with good compliance by both patients and controls. There was a concern that there would be a variation in frequency of movement; however there was only 4 occasions out of 32 studies when the scanning had to be halted in order to correct movement error. In the developmental work, headphones conveying the sound of a metronome (1 Hz) had been found to be too uncomfortable with the head coil.

It is worth noting that all the patients (n=16) were able to complete the protocol. However, due to the fatigue and pain in 4 of the patient groups it was not thought appropriate to undertake the further EMG studies. Further, two individuals, one from the patient group and one from the control group where excluded due to clinical findings on their MRI which may have affected the result. These individuals where recruited in addition to the 32 patients/controls, thus leaving 16 individual's in each group for the purposes of the analysis contained within fMRI results.

6.1.3 Discussion of the fMRI Results

6.1.3.1 All movement

One of the aims of this thesis was to development a protocol to measure cortical activation whilst undergoing fMRI and EMG.

The method development study used 4 healthy volunteers, which demonstrated activation in the appropriate areas (Table 3.2).

As discussed in Chapter 3.1.6, in the results section of the method development study data and in the literature review section, Chapter 2.2, the cortical activation is complex and variable. However, using

the method development study protocol, activations were seen in Brodmann Areas 5, 6, 7, 44 and 46.

It can be observed that activations occur throughout the analysis that is caused by cortical functions that do not relate directly to the research question. For example, activation in my analysis can be seen in Brodmann area 19. This Brodmann area relates to visual processing, which in the research paradigm would be activated due to the task, namely monitoring motor movement, and when the colour signal changed, indicating movement required. However, it is my intension to concentrate on activations that relate more directly to the movement, and not seek to analysis all areas of activation.

Overall, the activations set out in Table 4.4 are consistent with findings of published studies of hand and ankle movement [323-325], with activations in the primary motor cortex, sensory cortex and associated areas of the pre-motor cortex, supplementary motor cortex and the cingulated motor area.

As evidenced by Table 4.4 and Figure 4.9, predominant activation is in the left hemisphere, denoted by the –x in the MNI coordinate system, particularly in Brodmann areas 6, 13, 3, 7, 22. This is consistent with work on the transcallosal inhibitory system in relation to hand movement [326-330].

As can be seen the premotor cortex, Brodmann area 6, is the predominant area activated across all the movement of the controls. The role of this Brodmann area is set out previously, Chapter 2.2.2, being sub-divided into the supplementary, cingulated, lateral ventral pre-motor and medial ventral pre-motor area. In simple terms this is responsible for complex movement such as the movement of a joint [331], and as the paradigm being assessed was movement of the shoulder, this would be consistent with large activations in this area. Brodmann area 6 feeds into Primary Motor Cortex, which was also active but to a significantly lesser degree.

Brodmann area 6 receives inputs itself from areas 5 and 7, known as the Somatosensory Association Cortex, posterior to the postcentral gyrus. Brodmann area 5 is thought to contain a model of limb orientation and is involved in planning movement as well as inhibiting [158, 165]. Brodmann area 7, although linked with a speech role, has been linked also with movement [332-334].

Tanaka et al. has been shown that Brodmann area 6 has cognitive function, however, it was not thought this was a driving factor in the activations seen within the activations of this thesis [335].

There is a high level of activation of Brodmann area 13, a part of the insular cortex, which is part of the lateral sulcus; this marks the boundary between the temporal lobe with the parietal and frontal

lobes. The insular cortex has a number of functions, including verbal memory [336-338], pain processing [339-341] and in the left hemisphere phonological processing [342]. Beurze et al. established this area was responsible for reach planning, in conjunction with the posterior parietal cortex, premotor cortex and the medial frontal cortex [343].

Brodmann area 31 is the posterior section of the cingulated motor area; it is thought that this area is associated with computation of aspects of movement intention and movement monitoring [155, 344, 345].

Brodmann area 3, the primary somatosensory cortex, is responsible for the sense of touch [346]. Brodmann area 44 has many functions but relevant to our examination is the role for suppression of activation in the supplementary motor area [347].

Activation was also seen in the following areas which is ancillary to the task: Brodmann area 40 is responsible for reading; Brodmann areas 19 and 20 are visual center'; Brodmann area 37 has both visual and language functions; Brodmann 10 has a role in event-based prospective memory [348-365].

The inter-subject reproducibility of a research paradigm in an fMRI study is important in assessing the validity conclusion drawn from

the group analysis. One of the participants in the method development study was scanned again 13 months after the original scan (Table 3.9), which produced activations in both scans in Brodmann area 5 and 6. The t-value of the first scan of these two Brodmann areas ranged between 5.8-9.6 and for the second scan 5.71-10.21, all having p values <0.001 in Family Wise Correction.

6.1.3.2 Forward Flexion/Abduction

The results for forward flexion in the control group are set out in Table 4.5 and shown in Figure 4.10. Table 4.6 and Figure 4.11 illustrate the activations for the movement of abduction.

Forward flexion generates activations within the primary motor cortex, supplementary motor cortex, basal ganglia and cingulated motor area [155, 344, 345]. Activation in abduction was in the expected areas and similar to that in forward flexion. The number of activations is similar (Table 4.7) with 7 clusters for abduction and 10 for forward flexion if a voxel threshold of 10 is applied. If no voxel threshold is applied then there are 21 clusters for forward flexion compared to 20 clusters for abduction. However, a greater level of activation occurs for forward flexion (811 voxels) compared to abduction (305 voxels). It needs to be remembered that the range of motion for forward flexion was 30 degrees and 15 degrees for

abduction (Figure 3.9). The reduced range of motion explains the smaller voxel activation between the two movements.

However, when these two movements are subtracted as a contrast, there are no surviving voxels after the Family Wise Correction is applied. This result was consistent with the method development study, Chapter 3.1.5. This is of course testing for activations that are unique to either forward flexion or abduction. The absence of such unique activations is perhaps unsurprising on the basis of the following:

i. As set out in Chapter 5, there are many similarities in the muscles used to produce these movements, particularly within the confines of the scanner.

ii. In primates a single corticomotoneuronal cell may have multiple functional connections, to multiple muscles and multiple anatomical locations in the upper limb [142, 166]. Common corticomotoneuronal cells may have same role in both movements.

iii. Movement of joints has been found to induce a wide neuron activation in the motor cortex [167], which may be common to both movements.

6.1.4 Conclusion

The main study, which combines both movements in the analysis, demonstrated activation in the primary motor cortex, the supplementary motor cortex, the cingulate motor area and the primary somatosensory cortex. There was good reproducibility and thus it can be concluded that the developed paradigm was suitable for testing the movement of the shoulder whilst in a scanner.

It was not possible to distinguish between activations of forward flexion and abduction. Inclusion of these two different movements was warranted on the following basis:

i. It provides different variation and thus stops the subject using different areas of activation of learnt behavior [257];

ii. In a patient group with painful and dysfunctional shoulders, it reduced the load and thus increased the likelihood of completing the protocol task.

As we know from the data reported in Chapter 4.2.1.4, there was no difference in the cortical activations of the two movements. However, in other shoulder pathologies there may be such a

difference in activations and voxels survive, our protocol would reveal these differences in activations.

In terms of the activations of the control group alone, it can be seen that voxels are shown in the expected areas. It can be concluded that the developed fMRI protocol is appropriate to examine movement of the shoulder.

6.2 Comparison of actions – Patients versus Controls

6.2.1 Patient group selection

The method of selection is set out in Chapter 3.4.4. Of the patients with type II/III shoulder instability, the WOSI and OIS were used as pseudo-markers for the extent of their shoulder functional deficient (Figure 4.1 and 4.2). In terms of both functional scores, there was a significant difference between the two groups ($p=0.001$), as expected, but there was a wide spectrum of dysfunction within the patient group. This reflects the different stages of treatment and the fact that some of the patients either only achieve small improvements or are in remission.

The patient numbers as discussed Chapter 3.4.1 are small even though our Unit at the Royal Liverpool Hospital is the referral center for the whole of the North of England. It would have been advantageous to undertake the protocol on initial referral to the Unit and then systematically during treatment. Instead the patients in the study were at various stages of their treatment. However, the numbers who truly fall within polar type II/III is too small to enable an attempt to test the patients upon initial referral and achieve sufficient numbers in the study to mitigate against type 1 errors.

Beck's depression inventory (Figure 4.3) showed a significant difference (p=0.001) between the two groups. However, in both the patient group and to a certain extent the control group there is a wide spread of scores. The higher scores in the patient group are to be expected with a shoulder condition that is both painful and disabling.

6.2.2 Discussion of results

6.2.2.1 Patient all movement, forward flexion and abduction

In terms of movement related to activations, there are activations within the primary motor cortex (Brodmann area 4), the supplementary motor cortex (Brodmann area 6/4), the cingulated motor area (Brodmann area 31), the primary somatosensory cortex

(Brodmann area 3) [366] and the basal ganglia (putamen, ventral lateral nucleus).

When the movements of forward flexion (Table 4.2/Figure 4.5) and abduction (Table 4.3/Figure 4.7) are considered separately, the activations occurred in the expected areas. These differences between the patient group and control group in terms of Brodmann areas and levels of activation are considered in the next section.

As indicated previously, when the movement of forward flexion and abduction were subtracted there were no remaining voxels after Family Wise Correction. In the control group for the reasons previously set out, it is not surprising that there were no voxels that survived this comparison. As is explored in Chapters 5.3, 5.4 and 6.3, the EMG for the patient group demonstrates very dysfunctional muscle activation and patterning. It was thought this might translate into different activation between the two movements.

6.2.2.2 Patients versus Controls

6.2.2.2.a Voxel Activations

The fundamental question, the first question to be addressed in the thesis, was whether there was a difference in activation between the patients and the controls, Chapter 4.2.3.

The activations of all movement for the patient group are set out in Table 4.1, Figure 4.4, Table 4.4 and Figure 4.9 illustrate the activation for all movement for the control group.

When the control group collective activations (n=16) were subtracted from the patient group (n=16) and a Family Wise Correction at a voxel level, one voxel remains (2 mm x 2 mm x 2 mm) at MNI coordinates, -38 -26 56 (Figure 4.13 and 4.14). At a voxel level the group comparison, through the GLM based on random field theory modeled within SPM, produces this voxel with a t value of 5.22 at a Family Wise Correction p=0.04. The voxel has survived the very conservative and robust Family Wise Error correction area and around it there is a cluster of a substantial size, which is described in the next section.

This location is within the left hemisphere (which is denoted by the first coordinate (-38) being a negative number). The WFU Pick atlas

has the location as being the left post central gyrus. This area is the somatosensory cortex, which contains Brodmann areas 1, 2 and 3. However, using the coordinates through the WFU Pick atlas does not define a Brodmann area and observation places the coordinates at a point close to both Brodmann area 3 and 4.

SPM Anatomy Probability Atlas was used to estimate the functional area of the cortex, which demonstrated an 89% of being in the primary motor cortex (Brodmann area 4), and 11% chance of being within the somatosensory cortex (Brodmann area 3).

The MNI coordinate appears as part of work looking at the role of the anterior cingulate cortex in relation to coordinated motor behavior undertaken by Wenderoth et al. In this work the coordinate was activated in a task that required a high level of coordination [367]. The research paradigm undertaken in my study did not involve movement that required a high level of coordination. Given the simplicity of the task, I suggest the activation is more consistent with a compensatory strategy.

There has been a great deal of work looking at compensatory activation in stroke patients [368], preclinical Parkinson's disease [369] and Huntington's disease [370].

Grefkes et al. [279] studied improving motor function in stroke patients. They found that some of the motor deficiency is not related to the ischaemic lesion but relates to cortical regions distant to the lesion, presumably instead to the over inhibition of the contralateral intact motor cortex. This over-active inhibition has been established through DCM [371], demonstrating disturbances of the motor and supplementary motor cortex [372, 373]. In Grefkes et al. work between a base line fMRI scan and further fMRI scan three months later, patients underwent rTMS. The study showed that rTMS over the contralesional motor cortex was associated with increased function in the affected hand.

Grefkes et al. work provides assistance in advancing a theory to explain activation of this area in the patient group. The MNI co-ordinates between my study and that of Grefkes et al are the same. However, through his use of DCM it was concluded that the coordinate had an inhibitory effect in stroke patients. On my results it cannot be said whether this is inhibitory or increased activation related to a compensatory mechanism. Given a single voxel may be responsible for numerous functions, it could of course be a combination of both inhibitory/increase activation.

Further, the MNI coordinates (-38 -26 56) have been identified in work exploring the role of the basal ganglia during motor performance after demanding motor tasks undertaken by Bonzano

et al. [374]. The task was sustained opposition of the thumb to the rest of the four digits sequentially for a period of 2 minutes, defined as a demanding finger motor task. The above co-ordinate was the center of the greatest activation during this task. During the activation of this area, there was a compensatory increase in activation of the basal ganglia, possibly to compensate for motor performance deterioration due to a central fatigue. This is of relevance to the present results; one is that given the simplicity of the task this is evidence of an increasingly complex compensatory activation; another is that (similar to Bonzano's findings) there is a parallel circuit operating with the basal ganglia to compensate for a central fatigue condition; or, lastly, that the activation is merely coincidence.

Wenderoth et al. [367] found activation in this area when the motor task was more complex, hypothesising that this area had a modulatory effect on other motor areas; including the primary motor cortex and the supplementary motor area. Drawing on Wederoth et al. works' it reinforces the proposition that in my study the activation is suggestive of a compensatory activation.

As has been observed from the OIS and WOSI scores, the patient group reflects a spectrum of type II/III shoulder instability (Figures 4.1 and 4.2). Further inspection of the patient scores reveals a patient with a WOSI score of almost 0 and an OIS of 48, almost

identical to the control group. Further, examination of the two figures show that the rest of the patient WOSI and OIS were dramatically different to that of the controls. However, we know this individual patient was a polar type II/III, but at the time of testing exhibited little evidence of shoulder dysfunction.

A retrospective examination of the co-ordinate (-38 -26 56) in the patient with the effectively normal WOSI and OIS was undertaken in looking at their first level model. Figure 4.34 illustrates that this co-ordinate is not activated during the contrast of all movement compared to the rest. The same analysis was retrospectively undertaken for the entire patient group, an example is shown in Figure 4.35, which shows the activation at the co-ordinate. The activation at the co-ordinates was activated in all of the other patients at the first level save for the one patient who had in effect a normal WOSI and OIS.

6.2.2.2.b Cluster activations

Table 4.9 illustrates the five clusters that result from the second level GLM contrast controls subtracted from patients at Family Wise Correction of p=0.001. The six Brodmann areas that are included in these clusters are: area 3, Primary somatosensory cortex; area 4, Primary Motor Cortex; area 6, Premotor cortex; area 9, Dorsolateral prefrontal cortex; area 40, Wernicke's area; area 44, Broca's area.

The last two Brodmann areas, 40 and 44 can safely be ignored, as they are only ancillary to the task.

Figure 4.18 illustrates the co-ordinate (-38 -26 56) that survived at the voxel level, the top portion of the segment that straddles Brodmann areas 3 and 4.

The increased activations in Brodmann areas 3, 4, 6 and 9 is evidence either that the patient group is working harder to achieve the same movement or the activation relates to dysfunction inhibition.

These results are produced through a GLM, but are consistent with the independent second level findings for each group. Table 4.7 shows for all movement that the total level activation for the patient group was 3,783 voxels compared to 3,259 voxels for the control group.

Thus the motor representation in the cortex is able of be changed either by centrally driven changes, such as an ischemic lesion [173] or peripherally damage, such as in Leprosy. Conditions such as leprosy have found to increase motor cortex representations [375]. Leprosy is a condition where Mycobacterium peprae attacks the skin and the peripheral nerves which causes progressive motor, sensory

and autonomic dysfunction. This work would suggest a peripheral problem changing the central motor representation.

From the large volume of fMRI work related to stroke, set out in Chapter 2.2.4, the central changes result in both the sensory and motor cortex [193]. Although there are increased levels of activation, there are normally in the contralateral cortical hemisphere [194-196].

Wang et al. has shown an increase in activation in Brodmann areas 6 and 3 in patients with Multiple Sclerosis, this is thought to be a result of interhemispheric reorganisation of the motor area [376]. Ipsilateral increase in activation in the motor cortex has been reported in conditions such as action-induced dystonia in writer's cramp [377].

The increase in Brodmann area 9, the dorsolateral prefrontal cortex shows this area has a function in motor execution, planning, organisation and regulation. This area has also been shown to be involved in the execution of complex motor behaviour and involved in the recruitment of fronto-parietal networks and sensorimotor regions [378]. Thus the patients deploy cortical regions traditionally involved with complex motor tasks, even though the task is simple. This is at least suggestive of complex compensation.

6.2.2.2.c Comparison of Brodmann Areas

Figures 4.28-4.33 illustrate the number of clusters in the various Brodmann areas for both the patient and the control group. This data is based on the second level analysis of both groups individually, these activations having survived the voxel level Family Wise Correction <p=0.05.

In terms of the primary motor cortex, Brodmann 4, the patient group across all movements has greater activations in this area compared to the control group. The control group has a greater number of clusters in the supplementary motor cortex, Brodmann area 6. This reorganisation of motor function is seen in a number of central pathologies such as stroke and multiple sclerosis [379], however, the cause in the patient group must obviously be different.

The Figures 4.28-4.33 further show increased activation in the basal ganglia of the patient group. This is consistent with an increased activation at the coordinate (-38 -26 56) in our patient group and an increase in activation of the basal ganglia to compensate for motor performance deterioration in central fatigue.

6.2.2.2.d WOSI and IOS contrast

The two functional shoulder scores, WOSI and IOS were used as a convarient against all movement of both the patient and the control group. These results are shown Tables 4.11 and 4.12, along with the graphical representation in Figure 4.36 and 4.37. Activations were seen in Brodmann areas 3, primary somatosensory cortex; 6, supplementary motor cortex, 11, orbitofrontal area; 26, cingulate gyrus and the amygdala.

The findings with respect to the primary somatosensory cortex, supplementary motor cortex and the amygdala are consistent with the previous findings in the previous Chapter 6.2.2.2a, is suggestive of a compensatory activation and a parallel activation of the limbic system.

Activation in Brodmann area 11 is interesting as lesions in this area cause patterns of disinhibited behaviour, namely, poor social interaction, hypersexuality, swearing excessively, compulsive gambling and drug use [380]. The function of Brodmann area 26 is largely unknown although it has been linked to reward decision making [381].

6.2.3 Conclusions

There is clear fMRI evidence of a motor and sensory reorganisation within the patient group.

The increase in activation at the voxel level Family Wise Correction is powerful evidence that in the patient group there is additional activation within the motor cortex. This increased activation in the patient group at the cluster level encompasses the primary motor cortex (Brodmann area 4) and the primary somatosensory cortex (Brodmann area 3).

Drawing from previous work in neurological conditions of various pathophysiologies, it is well established that the cortex reorganisation is a compensatory strategy to maintain or improve limb motor function.

These observed differences in the fMRI activations are starting points from which future work will achieve better understanding of the difference in activations and use neuroplasticity to make noninvasive changes.

However, as Grefkes et al. [279] demonstrated, interventions such as rTMS can modulate the motor activation difference and improve

function. I suspect that this is what occurs when the shoulder instability patients undergo muscle-patterning physiotherapy.

This work establishes there is a difference in cortical activation in the patient group that relates to both the motor and sensory cortex. This objective finding will enable future patients to understand there is a difference in the manner they active their shoulder and why it dislocates. It provides information that can springboard recruitment of other patients into future studies.

6.3 Electromyography

This section will look at the muscle activations forward flexion and then abduction. First I compare activations in standing and supine position, then comparing patients and controls.

As set out in Chapter 2.3, the sum of our EMG knowledge derived from papers totals 60 patients in the simple movement of forward flexion and abduction in the standing position. This total number of patients is derived from a number of papers and closer examination of the work reveals smaller numbers for some of the individual muscles.

As previously set out, no work has comprehensively reported the results of muscle activation in the supine position.

6.3.1 Forward Flexion – Standing versus Supine

6.3.1.1 Anterior Deltoid, Middle Deltoid and Posterior Deltoid

Myers et al. [215] in forward flexion demonstrated, as one might expect, that anterior deltoid has greater activation than middle deltoid, which in turn has greater activation than posterior deltoid. This work, similar to our own, normalised the muscle activation to the MVC, and thus it is not possible to rank muscles in terms of greater or less activation. The character of the muscle activation of course changes with the degree of forward flexion. The work by Heuberer et al. [218], showed that in phase 1, the degree of slope was similar in AD and MD, with a shallower gradient comparing his work to my own results.

Figures A1.1, A1.3 and A1.5 illustrate that AD peaks slightly before MD and PD in Phase 1 in the standing position. In Phase 2 AD and PD are important in centering the humeral head as there is activation throughout this phase, where there is a general reduction in the activation of MD. It has to be remembered that in the standing role, some of the action of muscles is controlling movement against gravity. Thus in Phase 2, as the arm reaches 90 degrees in the downward swing, there is a peak of muscle activation of AD, which would arrest the descent of the upper limb. Towards the end of

Phase 2, there is increased activation of PD, bringing the upper limb to the side of the body.

It can be seen (Table 5.1) that the mean level of activation of all three muscles (AD, MD, PD) is moderately reduced in the supine position compared to the standing position, for example 6.3% less for AD.

In the supine position the change in gravitational direction is reflected in the muscle activation. As previously emphasised in the results Chapter 5.1.1, the range of motion test replicated that movement within the scanner. Forward flexion totaled 30 degrees and abduction produced a movement of 15 degrees. So if a comparison is being made of AD between the supine activation, Figure 5.4, compared to the standing position, Figure 5.3. AD, in the first 17% of Phase 1 in the standing position is equivalent to the whole 100% of the Phase 1 in the supine position.

In the supine position there is a more consistent activation of AD, MD and PD. There is evidence in Figure 5.4 that AD is resisting gravitational pull half way through phase 2. However, the character of PD is slightly different, with a peak occurring halfway through Phase 2 rather than at the end of Phase when the individuals are standing. Further, in the supine position, MD, in Phase 2, there is a

fairly consistent activation compared to gradual reduction in the standing position.

6.3.1.2 Upper Trapezium and Serratus Anterior

The origin of UT are the spinous processes of C1 to T12 and the insertion is the posterior border of the lateral third of the clavicle, the acromion, and the spine of the scapula. The EMG electrode was placed on the superior fibers, which are largely activated during elevation. In forward flexion SA rotates the scapula forward and upwards, which enables the elevation of the arm [382].

The character of UT is not dissimilar to AD in both Phase 1 and 2. In the standing forward flexion, the elevation is important to rotate the scapula and thus increase the angle of the glenoid, to achieve the last 45 degrees of forward flexion [383]. This role is demonstrated by the marked increase in activation at the end of Phase 1.

6.3.1.3 Biceps Brachii

As one would expect in forward flexion, activation is larger in Phase 1 compared to Phase 2. The character of the activation is similar in the two positions, standing and supine.

The three peaks of activations across the two phases are maintained although the intensities are slightly different.

6.3.1.4 Teres Major, Latissimus Dorsi and Pictoralis Major

The superior fibers of PM were measured, whose principal action is to flex the arm; the activation (Figure 5.17) in the standing position shows that this muscle's greatest activation of the arm was at 90 degrees and in the downward swing at a similar angle, although to a lesser extant. This second lesser activation resists gravity, enabling controlled descent of the arm.

LD and TM have a greater stablising role in forward flexion, with LD exerting a gradual tension on the scapula whilst the arm is rising. Further, in phase 2, when PM is controlling the descent by exerting an upward force, there is a corresponding activation in Phase 2 by LD. TM has a stabilising role between the scapula and the humerus in an inferior direction preventing superior translation during forward flexion, as has been noted in other studies [218]. The peaks in Phase 1 of TM and LD indicate a more gradual tension, lagging behind the some of the primary flexors such as PM and the deltoids.

The results for all three muscles confirm that there is good activation in the supine position, although the character of the activation is

different to that in the standing position. As is to be expected the activation over smaller angles is smaller.

6.3.1.5 Supraspinatus, Infraspinatus and Subscapularis

SSP elevates the humerus as can be seen in our findings, consistent with previous work. It has been shown that SSP has a complementary role with that of AD, [384]. The muscle patterning shown in my study for both SSP and AD is identical to this previous work. The peak of AD occurs after the peak of activation of the SSP. This latency between these two muscles is preserved in the supine position although due to the reduced range of movement the peak is shallower in the supine position.

In the coronal plane, Inman et al. [229] describes the muscles of the inferior cuff, namely SUB, ISP and Teres minor, resisting the superior migration of the humeral head induced by the deltoid muscles. This coupling is evident in the activation data (Figure A1.1, 5.23, 5.25), with SUB and ISP activating prior to the AD peak, plateauing during the AD activation, then reducing once the AD activation intensity has decreased.

In the inferior rotator cuff the SUB exerts an anterior force and ISP a posterior force [385]; the shape of the activation curves (Figure 5.25 and 5.23) demonstrates the inferior-anterior-posterior coupling.

For the coupling described above for AD, ISP and SUP in the supine position there is limited evidence can be seen when comparing the activation curves. This could be due to the fact that the movement is over a small range of motion and the gravitational action is at 90 degrees compared to the standing position. In the supine position the coupling is more subtle as the activation of these muscles is not as pronounced compared to standing. However, in phase 1, there is an activation of both SUB and ISP prior to and proportionate to AD. The evidence of the coupling relationship is demonstrated by similarity of the curve amplitudes.

ISP and SSP stabilise the shoulder in forward flexion [386]; in particular during Phase 1 it can be seen that ISP exerts a consistent activation which, given the location of origins and insertion, generates a constant antagonist force to the main flexors. This tension across the joint prevents translation of the humeral head across the glenoid. For both of these muscles, although the activations in the supine position demonstrate good activation, there character is subtly different to activation of forward flexion in the standing position. However, the activation curves of ISP and SUB mirror the amplitude curves of each other, although the curves as expected are of a different character compared to the standing position.

In forward flexion, SUB counterbalances the forces exerted by ISP in the axial plane and provides dynamic stability across the glenohumeral joint [387]. Comparison of the activations in the standing position of these two muscles confirms this analysis.

In the supine position although the character of the activation is different, it can be seen that there is a similar pattern, which would be consistent with the ISP acting as antagonist to SUB.

6.3.2 Forward Flexion – Patients versus Controls

When trying to make sense of the results it is important to remember that structurally the shoulder is largely intact. This is different to other shoulder pathologies, where the compensatory strategies arise from structural abnormalities, as in rotator cuff injuries.

The patients' level of activation is high comparing the 10 muscles to that of the controls (Table 5.3 and 5.4). The patient group in phase 1, range from 86-112%, and phase 2, range from 78-103%. This compares to the control group, who in phase 1 range from 38-73% and in phase 2 range from 8-71%.

Comparing the upswing and the downswing, there is no statistical different in the patient group between any of the muscles, compared to half within the patient group. This is testing the movement of

forward flexion to 90 degrees, in the control group there is a significant difference in half of the muscles. Thus it can be concluded that the muscle activation of the patient group is less distinctive between the two phases compared to controls.

The peak amplitude and where it occurs within the phase can be used as a marker to compare muscle patterning between the two groups. In Figure 5.9 and Figure 5.10 comparison between the two groups shows a difference in timing of the muscles ($p=0.012$). It can be observed that comparing the two groups there is no simple patterning.

The two graphs demonstrate that the activation in the instability patients is complicated. A number of previous studies [81, 83, 388] have suggested that single muscles are responsible for the instability, which on the basis of my results is too simplistic. There has also been inconsistency in research between the culpable muscle leading to the instability [80, 85, 93, 239, 250].

Subjective observation of the muscle patterning of the ten muscles reveals a profound difference in the manner of activations. The patient group activations show much greater variation within the general trend of activation levels. Namely, that the control group demonstrates consistent rates of changes in amplitude (producing smooth graphical representations), whereas, the patient group

shows erratic changing levels of amplitude within the general trend (producing erratic graphical representations).

The shoulder is dependent on synergistic activation of both agonist and antagonist muscles to achieve movement while maintaining the humeral head centrally within the glenoid. The random variations of activations can easily be appreciated as leading to difficulty in maintaining the humeral head within the center of the glenoid.

Thus on initiation of movement, the muscles of the shoulder endeavor to maintain the humeral head in the center of the glenoid. The comparative erratic muscle amplitude patterns of the patient groups demonstrate the disorganised manner in which this is achieved. A dynamic chain reaction between the muscles in the patient group means:

i. That agonist and antagonist muscles are forced to respond to the disorganised activations, which lead to a constant state of erratic activations.

ii. That in order to maintain stability greater amplitude is required, which exacerbates the need for constant readjustment between coupled muscles.

6.3.2.1 Anterior Deltoid, Middle Deltoid and Posterior Deltoid

The main difference between the patients with the controls is the level of activation is greater at the start of the movement, the peak is less defined and there is a secondary peak in Phase 2.

The level of activation in the patient group results in increased activation in the coupling muscles. For example ISP and SUB resist the superior translation of AD. Thus comparing the activations of AD (Figure 5.11) and ISP (Figure 2.19) the increased baseline activation has a similar pattern of amplitude. In this part of the study SUB was not tested, as it was felt the patient group would not tolerate the insertion of the fine wire electrodes.

The peaks of activation are biphasic compared to the smooth gradual peak in Phase 1 (Figure A2.3 and 5.13). This type of activation has been observed in ankle instability [389].

The biphasic activations observed in AD other muscles of the patient group are of greater variability and consistent with an over activation of the sensory motor cortex as explored in 6.2.

6.3.2.2 Upper Trapezium and Serratus Anterior

The activations of UT in the control group are lower but consistently elevated in the patient group, which indicates a compensatory mechanism. The upper fibers of UT are responsible for elevation and forward rotation of the scapular [390]. In the control group the amplitudes suggests rotation and elevation of the scapular, whereas for the patient group the amplitudes suggests it remains fixed or under held under tension from UT.

In forward flexion in movement from 0 to 60 degrees the movement is predominantly achieved through the gleno-humeral joint. It is between 60-120 degrees that the scapula moves forward, enabling the glenoid surface to point upwards. Activation of UT (Figure 5.15) suggests the scapula is rotated forward early in the movement cycle in the patient group.

SA also is activated at a lower level and the activation is different in character to the control group. In particular there is a prominent peak present in Phase 2, present in other muscles (Table 6.1). There is a shallower peak in Phase 2 in UT also. This activation could either be compensatory or a provoking factor that causes other muscles to be develop a defensive compensatory activation to endeavor to maintain glenohumeral stability. The inability to explain this phenomenon is a limitation of the EMG methodology. Given the

dynamic process of shoulder movement, I would suggest it is a combination of both provocation/compensation throughout the various muscles at different points in time.

6.3.2.3 Terres Major

This muscle in the controls (Figure A2.12) antagonises the main forward flexors, providing a consistent tension, with little variation of activation.

The patients have a more fluctuant activation (Figure A2.11) and there is a biphasic peak in Phase 2, similar to AD, MD, UT and SA. It is suggested that these activations will cause the translation of the humeral head on the glenoid to be greater and less consistent.

6.3.2.4 Pectorialis Major and Latissimus Dorsi

Overactivation of PM and LD has been suggested as a cause of shoulder instability [243, 292]; however other studies showed differences only in PM [244]. Studies of upper limb movement in stroke patients has shown abnormal activations of PM in both forward flexion and abduction [391].

There is greater mean activation in Phase 1 in the patient group, LD 111%, PM 120.9 compared to the control group, LD 97%, PM 100%

(Table 5.6 and 5.7). In Phase 2 there is a great mean activation in the control group, LD 101%, PM 97% compared to the patient group, LD 90% and PM 81%. Thus there is over-activation in Phase 1, which would agree with the previous cited work [90]. However, to attribute the instability to one or more muscles or one or more phases is over simplistic. What these result show is that there is a difference in activation in both phases and this may cause either actual or perceived instability.

LD in Phase 1 shows a similar activation pattern although there is greater variation. However, in Phase 2 a biphasic peak occurs in the middle of the phase compared to the controls. There is a later peak in the controls but the level of intensity is smaller.

The character of the activation of PM is different between the two groups. The patient group has lower levels of activation and a peak in Phase 2, which corresponds to a declining activation in the control group.

6.3.2.5 Bicep Brachii and Infraspinatus

The role of BB in forward flexion is one of the primary flexors in the range of motion of 0-90 degree, and in the downswing resists gravity and maintains glenohumeral stability [390].

The activation in the patient group during Phase 1 has a peak that is of different character to the control group, which shows a more gradual onset. In Phase 2 there is an additional biphasic peak that is absent in the control group. The peak for the patient group occurs approximately 70% through the phase 2, demonstrating that BB in involved in maintaining a downward force on the glenoid. This increased level of activation may represent a distracting force causing instability or a compensatory strategy to maintain stability.

The peak in Phase 2 in respect of BB and ISP, is similar in occurring approximately 70% through the movement cycle (Figure A2.17 and A2.19). The similarity would be consistent with the coupling action of these muscles given their anatomical location and the similarity of activity in this phase.

6.3.3 Abduction – Standing versus Supine

In the previous section, Chapter 6.3.2, considering the movement of forward flexion a number of generic observations that applied to all movements of the shoulder were made. It is not intended to repeat observations or conclusions but to focus on specific conclusions that relate to abduction in both the standing and the supine position.

In particular, the activation patterns in the supine position for all the muscles tested demonstrated activation, on the whole at a higher

baseline compared to that of the standing position. However, the character of the activation in the two was often not similar. For the fMRI protocol it is the activation that is important and there is sufficient similarity to make the protocol valid. In the interests of economy this point will not be made throughout all the discussions of individual muscles. So for example, if the amplitudes of AD (Figure 5.21) in the standing and the supine position are compared, in the standing position there is a definite peak in phase 1, with a secondary smaller peak in phase 2. However, in the supine position (Figure 5.22) the peaks look different in character, in part due to the decreased range of motion, however, there is a constant amplitude (mean 86-117%). Thus in the supine position there is good level of muscle activation.

In the standing position, (Table 5.5) the difference between the muscle activation in Phase 1 and 2 was significant ($p=<0.005$ to 0.027). This contrasts with the supine position (Table 5.6), where there was no statistical difference in PD, LD, PM and SUB but significant difference in the remaining muscles ($p=<0.001$ to 0.007).

What this establishes is that testing shoulder movements in the supine position are representative of standing shoulder movement. As with forward flexion and as will be explored for abduction, there are subtle differences. In terms of fMRI paradigm there are

sufficient similarities in both activations to conclude that it is a valid protocol, but this limitation needs to be appreciated.

6.3.3.1 Anterior Deltoid, Middle Deltoid and Posterior Deltoid

These three muscles, along with SSP are the prime adductors of the shoulder [93], with Yu et al. concluding that MD was the chief adductor [392]. Given that each muscle was standardised to the maximum activation, intermuscle comparison of activation is not possible. What can be appreciated from Figure A1.19, A3.3 and A3.5, which illustrate activation patterns of AD, MD and PD, is that, as expected, the majority of activation for all three muscles occurs in Phase 1. Further, there is a secondary lesser peak in all three in Phase, which might be assumed to arrest the descent of the upper limb. However, in a small peak it is also present in the supine position, where the effects of gravity have been eliminated. Thus it could be concluded that as well as resisting gravity in the supine position, in addition there is an element of superior tension supplied by AD, to maintain a centered humeral head.

Because of the difference in their origins and insertions, all three muscles have the capacity to pull the humeral head in different directions. For example, in abduction MD generates external rotation torque that is counterbalanced by the contraction of the internal rotators SUB, AD, LD and PM [392]. It can be observed

from the standing activations that the biphasic activations in Phase 1 is counterbalanced by MD and PD to produce abduction, before the peak activations of LD and PM occur; this is consistent with the findings of Yu et al. [392].

In the supine position gravity is removed and the range of motion is limited to 15 degrees; however, there is greater activation in the peaks in Phase 1 (Figure A1.20, A3.4 and A3.6). The baseline activation in the supine position is greater but the character of the activation is different to that in the standing position.

6.3.3.2 Upper Trapezium and Serratus Anterior

As with forward flexion there pattern of activation of UT is similar to that of AD. The activation of UT in Phase 1 produces upward movement of the upper limb and resisting gravity. In Phase 2, there is a secondary lesser peak that allows the downward swing to occur in a controlled manner.

SA fixes the scapula until 90 degrees when the greater tuberosity of the humerus path is obstructed by the glenoid labrum and the coracoacromial ligaments. Then SA rotates the scapula in a forward direction permitting movement from 90-150 degrees, with lateral flexion of the vertebral column to the contralateral side to achieve the last 30 degrees.

Similar to forward flexion the supine activation of these muscles was different to the standing position, but there is sufficient muscle amplitude to be confident that the muscle is being activated during the fMRI protocol.

6.3.3.3 Bicep Brachii

Figure 60 illustrates that BB is instrumental in achieving abduction in towards the end of Phase 1, this contradicts previous recent work by Heuberer et al. [218]. However, given the origins and insertions, towards the end of the upswing, contraction of BB stops superior migration of the humeral head.

6.3.3.4 Teres Major, Lattisimus Dorsi and Pectoralis Major

As previously discussed LD and PM have a coupling antagonist role to MD [392].

The data demonstrates (Figure A3.15) high activation of PM during abduction, the literature being inconsistent as to the level of activation during this movement [215, 218, 229, 242]. Heuberer et al. [218] suggested a high level of activation indicating that it acts as agonist in abduction, which contrasts with Kronberg et al.[85, 93, 239] work suggesting the reverse.

The activation in the standing position (Figure A3.13) of LD is demonstrated, similar to other work, and the activations is essential to maintain shoulder stability [292]. Showing a high level of amplitude in phase 1 with a secondary small peak in phase 2. However in the supine position the pattern is very different, with there being a relatively constant level of amplitude at around 100% throughout both phases.

Figure A3.13 demonstrates the stabilsing role of TM in maintaining humeral head onto the glenoid, in particular after the upper limb passes 90 degrees in Phase 1. The role in adduction is demonstrated in Phase 2, with strong activations towards the end of the phase. Given the position (lower lateral edge of the scapula and the medial lip of the bicipital groove on the anterior surface) the contraction of TM completes adduction, bringing the upper limb in contact with the thorax.

6.3.3.5 Supraspinatus, Infraspinatus and Subscapularis

The coupling described by Inman et al. [242] between the deltoids (Figure A1.19, A3.3 and A3.5) and the inferior cuff, SUB and ISP (Figure A3.21 and A3.24) is demonstrated in the activation patterns seen.

In the standing position the complementary role of SSP and AD is not as pronounced in abduction compared to forward flexion. The amplitude for AD (Figure A1.19) compared to the activation of SSP (Figure A3.19) demonstrates little similarity.

At best the activations of these three muscles in the supine position shows activation but the similarity to the standing position is minimal. All of the amplitudes (Figure A3.20, A3.22 and A3.24) in the supine position for ISP, SSP and SUB show a level of action around the 100% mark.

6.3.4 Abduction – Patients versus Controls

There was not much difference in mean activations between the patients and the controls, which is in contrast to forward flexion, which showed greater activation in the patient group.

Comparing the difference in activations between the up and down swing, there are significant differences ($p=<0.001$ to 0.044), save for TM and LD the controls, with TM, LD and SA in the patient group. Given that the range of motion is 0-90 degrees, these muscles have a largely stabilising role.

Using the peak activation and its location within each phase, there is a greater divergence between the patient and control group in

abduction (Figure 5.24 and 5.25) compared to forward flexion (Figure 5.9 and 5.10). The only muscle that has a similar timing of peak activation is TM in Phase 2.

As with forward flexion there is no particular pattern between the two groups. However, in Phase 2 there appears to be a pattern (Figure 5.25), with patient activation occurring later than the controls for the majority of muscles. Although, when the anatomical origins/insertions are considered the pattern is not meaningful in terms of logically making sense of the pattern. What these figures demonstrate is how far the muscle activation is from normal. In a joint that derives its stability primarily from the muscles [73] the patients' activations difference can readily be appreciated.

From the testing undertaken it is not possible to establish whether these are compensatory strategies in response to dysfunctional activation of one or more muscles or whether there is overall dysfunction in all the muscles.

6.3.4.1 Anterior Deltoid, Middle Deltoid and Posterior Deltoid

Overall for all three muscles the variation in and around the general trend is less that observed in other muscles. The peak activations in Phase 1 have a more 'plateau' character and a lesser level of activation in the patient group.

In addition there is also a secondary lesser peak of activation in AD that is absent in the control group, similar to other muscles shown in Table 6.1. These sudden increases in activations in Phase 2 are matched by the coupling of the inferior cuff, shown by ISP (Figure 98). If this coupling is maintained then stability is maintained, although the effect is two fold. Firstly, the shoulder is working harder to maintain stability that will increase fatigue [393]. Second, the movement of the humeral head is more erratic and thus inducing either an awareness of instability or an actual instability. This would coincide with clinical experience of these patients both in terms of physical observation of movement and patient relating their experience of their affected shoulder.

Table 6.1. Table to show the muscles in the Patient group that have a secondary peak in Phase 2 of increased activation, which is absent in the Control Group.

Movement	Muscles demonstrating a peak in Phase 2 in the patient group that is absent in the control group
Forward Flexion	AD, MD, PD, UT, SA, TM, LD, PM, BB, ISP
Abduction	AD, PD, SA, TM, PM, BB, ISP

6.3.4.2 Upper Trapezium and Serratus Anterior

For UT activation of the patient group (Figure 88) has a 'plateau' nature, with less activation, which means that there is greater demand on the other primary abductors in Phase 1. For Phase 2, there is maintenance of activation in the patient group and a secondary subtle peak of activation towards the end of the phase. Thus to maintain stability in the shoulder in the downward swing the muscle is either having to work harder or its activation is causing other muscles to compensate.

The activation of SA in the patient group is very different, with a later activation in Phase 1, and additional activation in Phase 1, with overall a great level of activation. The character of the activation overall is higher in phase one which is unexpected as SA is more involved in abduction above 90 degrees. This is suggestive of the need to change the angle of the glenoid to achieve the same movement to that of the controls. The cause of the two activation peaks in Phase 2 is difficult to explain, other than this is further evidence of the discordance in the muscle patterning compared to the controls.

6.3.4.3 Teres Major

The controls (Figure A4.12) show the antagonist role of TM in both phases maintaining the humeral head in the glenoid, in Phase 1, enabling the glenoid to be used as a fulcrum by the primary abductors.

The patient group shows a similar pattern but the timing of the peak activation is different. Perhaps of interest is the double peak in Phase 1, rather than a gentle increase in gradient, the first peak occurring at 20% of Phase 1 cycle. It is not possible to be definitive on the effect this would have upon the humeral head position within the glenoid, but first principles would suggest that this may increase the translation compared to the controls or force agonist muscles to react to maintain stability. Note that AD and PD show an increase in activation at 20 percent of Phase 1 cycle.

6.3.4.4 Pectoralis Major and Latissimus Dorsi

There is a difference in the character of activation for both of these muscles between the patients and the controls. In Phase 1 activation of LD is greater in the patient group but the same for PM. In Phase 2, mean activation is greater in LD for the controls and in PM greater in the patient group.

Subjectively, the character of the activation is very different in both phases across the two different muscles.

6.3.4.5 Biceps Brachii and Infraspinatus

Previously BB inactivity has been implicated in shoulder instability [394] and the patient group in Phase 1 demonstrate less mean activation but greater mean activation in Phase 2 (Table 5.7 and 5.8).

However, the comparison of the character of the activation demonstrates the abnormal activation across both phases (Figure A4.17 and A4.18).

The activation of ISP is profoundly different with a great variation around the trend, with a secondary less intense peak of activation in Phase 2. This large comparative dysfunctional activation, given the stablising role of the ISP, is probably part of the explanation of why these patients experience instability.

6.3.5 Conclusion

The work demonstrates that the shoulder muscle activations in a normal shoulder are complex. It is important to consider the

character of the activations of the various muscles to truly understand their dynamic nature.

In the normal shoulder group the comparative activations in the standing compared to the supine movement, shows activation, the character of which is different in the majority of muscles. The activations in the supine position are sufficient for confidence in the fMRI paradigm. However, as shown in the difference in the character of activations, the assumption that they are identical should not be made.

The patient group as might have been predicted have a dysfunctional activation pattern. Previous work has cited individual muscles as being responsible for this dysfunction; my work demonstrates that this is too simplistic. In patients with Polar II/III instability there is not necessarily any single defect that generates the instability and thus one cannot conclude that muscle patterning is a compensatory mechanism.

The movement of the shoulder in a stable manner is a combination of muscle agonist, antagonist and synergy, and thus a defect in one muscle may start a chain of dysfunction of muscle pattern in others.

In the patient group in both movements the activations overall are higher, and thus they have to work harder to maintain stability.

Additional activations in the downward swing may cause additional humeral head excursion within the glenoid, requiring compensatory activation to prevent further instability and/or dislocation.

6.4 Limitations of Study

6.4.1 Generic

In common with most research studies you would like to exam all those in the patient group to ensure that the results represented the patient group accurately. This type of approach is not feasible from an ethical perspective or a resource perspective. Further, given the validation work surrounding the statistics/modeling used during the course of the study it would be unnecessary. However, although it is believed the study size of 16 patients and 16 age-matched controls was sufficient statically, it would be reassuring.

The muscle selection also identifies another common dilemma in research, the desire to simply test every variable in order to ensure that no influential/causative factor is missed. However, there needs to be a more disciplined approach based on pre-testing notions/ideas, in our case clinical suspicion. Without a disciplined approach there is a real risk that a research project descends into a random fishing exercise.

The reproducibility study was limited to one individual, again in keeping with most studies, it would be nice to retest all the individuals after a lapse of time to be confident the original testing represented a true picture. This approach again at both ethical and resource level is impractical, however, it has to be accepted that greater numbers including patients and controls would have been beneficial.

6.4.2 Study Specific

The patient group suffers from a condition that is difficult to categorise and is a continuum disorder, and so variation is inevitable in the patient group. Although using validated scores of instability enables a certain degree of consistency this is a self-reported score. Further, objective assessment of instability is problematic and in the testing session would, we felt, inevitability lead to some patients being unable to complete the fMRI protocol. Further, given the small numbers of these patients there was a variation in the stage of treatment and success of treatment.

We also choose based on the research set out at 2.2.3, to ignore the lateralisation of the subjects/controls. It would have been an interesting to test both upper limbs of both groups and then compare the activations to be confident that our approach was valid.

As mention, we cannot be confident that what the fMRI or the EMG is causative of the instability or a compensatory strategy to maintain stability. The increased activation could be cause the instability by activating the muscles in a disorganised manner. The misfiring of one muscle in one part of the movement would set up a chain reaction, causing the muscles and cortical activation to sense then correct the instability. It could be understood that if the misfiring was random during the movement cycle then there would have to be a rapid correction, which may be disorganised and set up further instability that need to be compensated for by further rapid correction. What is required is for the patients to undergo an fMRI and EMG immediately on diagnosis and then periodically through treatment (physiotherapy and then perhaps surgical) to assess the changes in both cortical activation and muscle patterning.

It was not possible to undertake a psychological assessment. From clinical experience it is felt that most of these patients have a psychological component to their condition, however, it was not possible to assess this aspect. None of the patient's has a psychological diagnosis; otherwise they would have been excluded from the study. However, for example a recently a patient who was part of the study and had been treated for in excess of 5 years, suddenly disclosed for the first time that she had been gang raped. This individual has been referred to a psychiatrist to see if there is any psychological illness.

The shoulder has six degrees of freedom of movement, thus in both the fMRI and EMG studies there may be variation in movement paths between subjects. The participants were closely observed to ensure compliance; however, there is no objective assessment.

In the scanner there was a limited range of motion, although we can be confident that there was muscle activation, the character of activation comparing the standing and supine position was different. Further, the movement itself is simple; it has been shown that abnormal activation within the motor cortex may only become evident with more complex tasks [395-400].

6.5 Suggested Further Work

In work looking at compensatory mechanisms in pre-clinical Huttingdons patients, Koppel et al.[401] has suggested that a shift in activations may be too simplistic, and that in addition to the activation there may be recruitment of additional areas. Scheller et al. developed this research into Huntington's disease using single Dynamic Causal Modeling ("DCM"). Through DCM a large number of connections were established that correlated with the severity of the pathological process and the complexity of the task being performed (for example, faster or more complex tasks induced connectivity alterations toward the pre and caudal supplementary

motor areas). The work in this thesis establishes some solid baseline findings in a patient group not previously studied using fMRI. However, DCM analysis might reveal further differences between the patient and control group.

A long-term project to assess patients before treatment starts and then periodically through their treatment would give great insights into the changes in both cortical and muscle activations. This work might enable conclusions to be drawn as to the causative factor in this type of instability. It has been demonstrated that passive movement training in healthy volunteers causes cortical changes in the primary sensorimotor and supplementary motor area [402].

It is the author's intention to set up a long term cross sectional study of Polar II/III patients'. The aim would be to undertake a baseline fMRI and EMG study on initial contact with the patient and periodically through their assessment and conservative treatment. It is thought this type of study would give powerful data that would enable determination of whether the cortical activation is provocative or evidence of neural compensation in response to peripheral neural dysfunction/muscle activation.

Although a robust protocol and analysis has been developed, it is believed that with the use of computer modeling of the controls versus the patient may give great insight into the EMG data. The

study demonstrates that it is difficult to characterise the movement through single figures, such as when the maximum amplitude occurs. Further, although comparison of the character of the amplitude of muscles provides more useful information, comparison is subjective and observational at best. The use of modeling, such Movement Deviation Profile [403], will enable great insight into the differences in the muscle activations of the patient group compared the controls. This method enables quantifiable comparison of complex data in multiple dimensions, which has previously been used in the analysis of gait data, Figure 6.1.

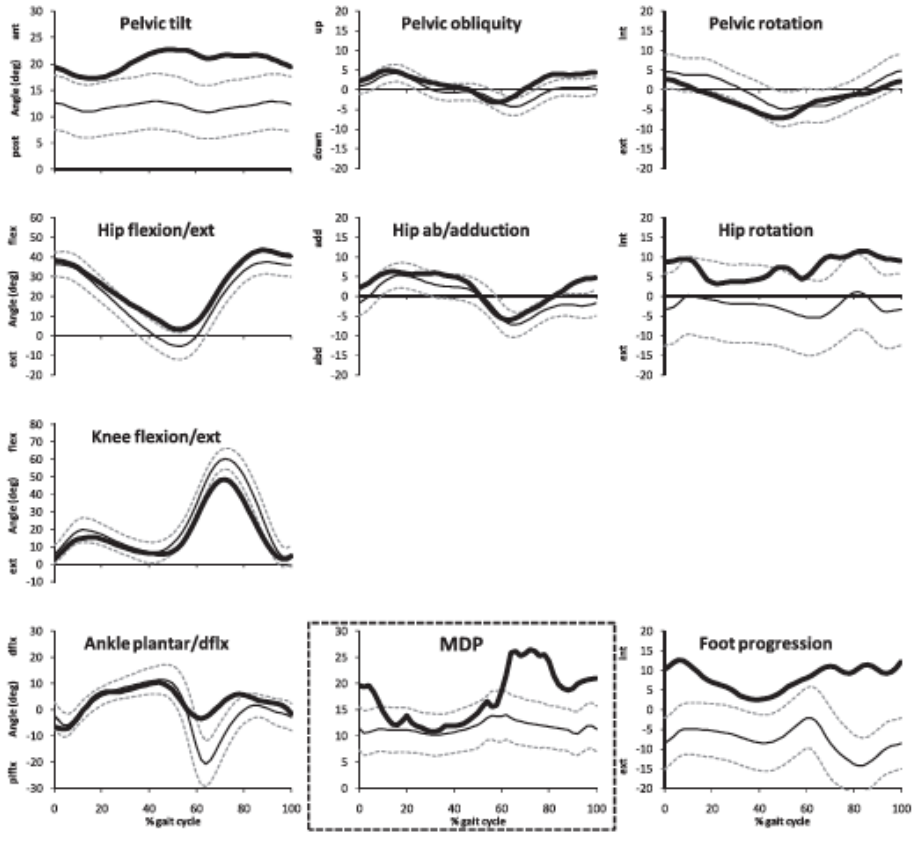


Figure 6.1 – Graphs to show the integration of Movement Deviation Profile in the conventional presentation of gait kinematics. The Movement Deviation Profile chart (surrounded by a doted line) summarises the other 9 angle curves of a patient and shows the timing of deviation from normality (movement deviation profile of controls with standard deviation bands) during the gait cycle.

The results of the findings need to be disseminated to both patients and medical personal. The patients who all within this group have often been poorly served by the medical profession, as their regular attendance with shoulder dislocation and an inability to explain/treat the cause as often leading to clinicians labeling them as attention seeking/psychological defective. Knowing there is an objective difference in the patients cortical activations will enable a greater empathy to be given to these patients from medical personnel. Further, from the patient's perspective knowing there is a rational explanation for their dislocation will in my view promote a great engagement with treatment rather than the buying into the idea that there is something psychologically wrong and thus their prognosis is hopeless. As mentioned previously it may be that there is a psychological component to the Polar II/III condition but again this will need to be factored into further investigations of the condition.

6.6 Overall Conclusion

My work demonstrates the there is a profound difference in both the sensorimotor and muscle activations of the patient group.

There is increase in cortical and muscle activations which is consistent with the notion that the patient group are in some sense working harder to maintain shoulder stability. However, it is not

possible to conclude when this is a compensatory strategy in the face of one muscle or whether the dysfunctional muscle extends to all the muscles are causative of the instability.

Given that there is additional activation in the motor cortex it is limited at a voxel level, I would suggest the dysfunction is restricted to part of the muscles involved in shoulder movement and the rest of the dysfunctional patterning is a compensatory strategy.

There are potentially far-reaching clinical implications for this group of patients. This group is often poorly treated, as healthcare professionals are unable to explain the recurrent dislocations and instability. For the first time I have demonstrated objective differences in cortical and muscle activation. This will help to dispel the myth that these patients are inducing their instability and/or dislocations. As medical professionals are unable to explain the recurrent dislocations, unfairly in the past these patient's dislocations have been felt to be a result of a psychological condition or attention seeking behavior.

Further, the rehabilitative approach to these patients has usually focused on one muscle or group. The EMG work demonstrates this is too simplistic and suggests that a more holistic approach needs to be adopted to the whole kinetic chain in shoulder movement.

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Appendix 1 – EMG –Normal Shoulder – Forward Flexion

Anterior Deltoid

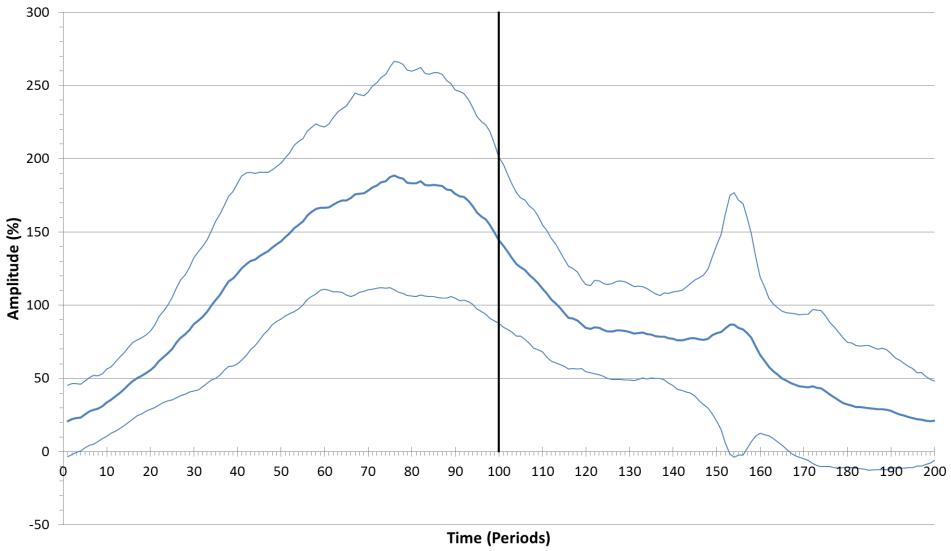

Figure A1.1. Graph to show normal shoulder group (n=19) activation for AD for the movement forward flexion. The thick and thin lines represent the mean amplitude and SD (+/-) respectively. The time period 0-100 represents 0 to 180 degrees in the upstroke and 100-200 represents 180-0 degrees in the down stroke.

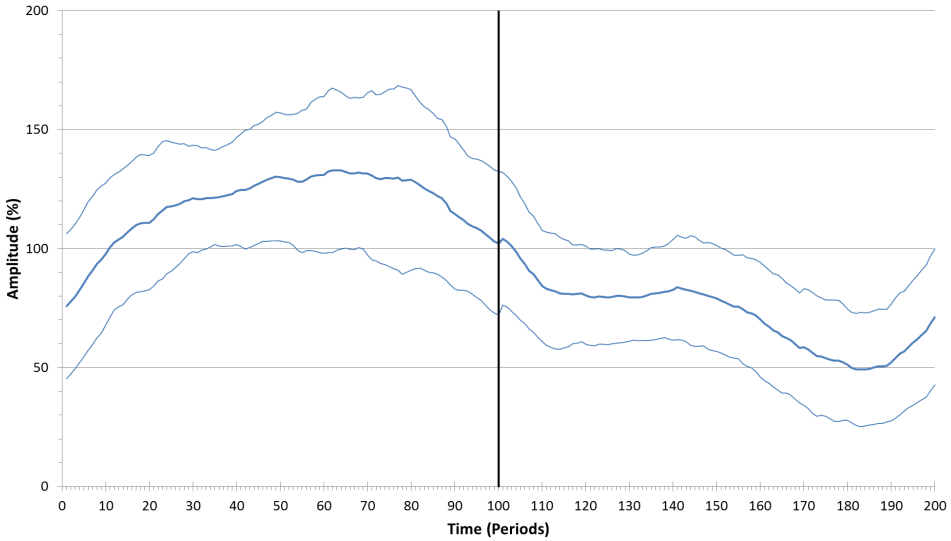

Figure A1.2. Graph to show normal shoulder group (n=22) activation for AD for the movement forward flexion whilst in the supine position. The thick and thin lines represent the mean amplitude and SD (+/-) respectively. The time period 0-100 represents 0 to 30 degrees in the upstroke and 100-200 represents 30-0 degrees in the down stroke.

Middle Deltoid

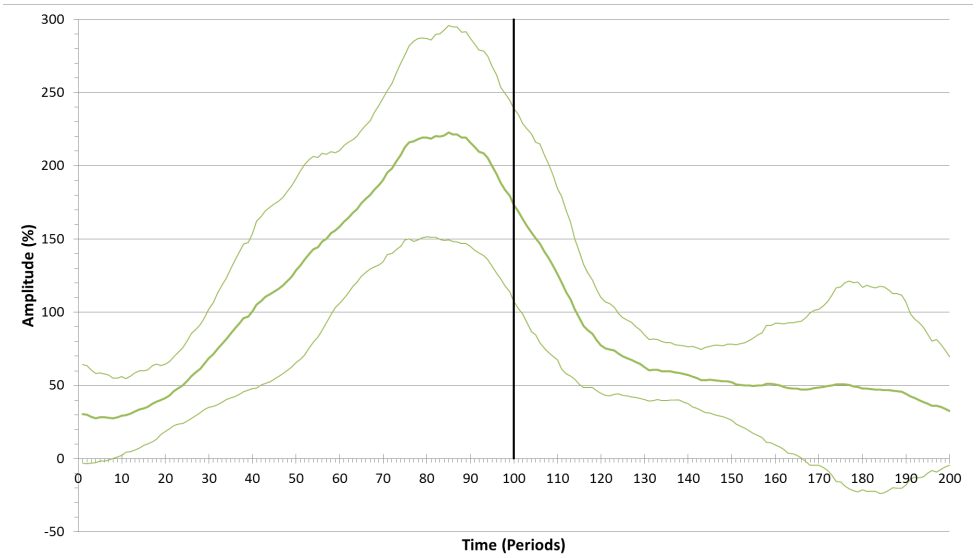

Figure A1.3. Graph to show the normal shoulder group (n=18) activation for AD for the movement forward flexion. The thick line and thin line present the mean amplitude and SD (+/-) respectively. The time period 0-100 represent 0 to 180 degrees in the upstroke and 100-200 represents 180-0 degrees in the down stroke.

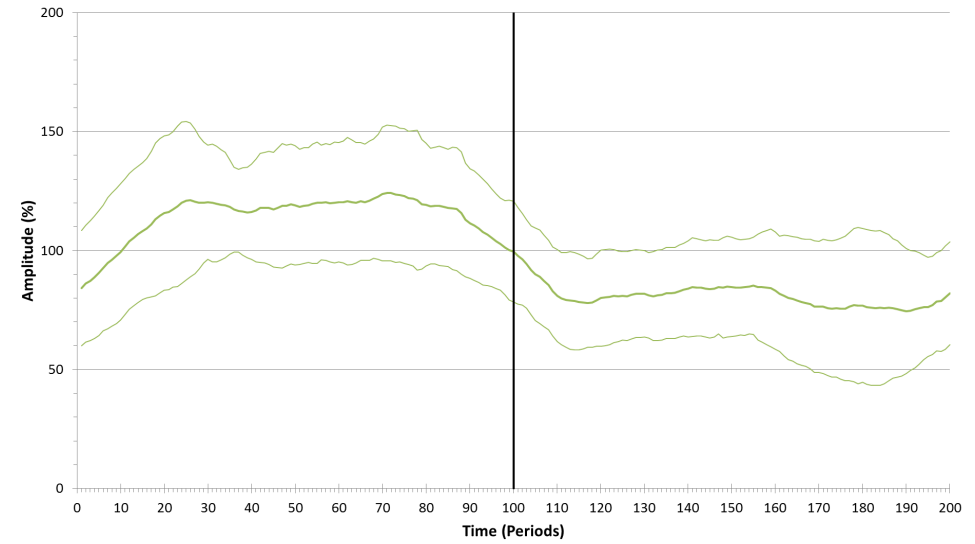

Figure Ap1.4. Graph to show the normal shoulder group (n=24) activation for MD for the movement forward flexion whilst in the Supine position. The thick line and thin line present the mean amplitude and SD (+/-) respectively. The time period 0-100 represents the upstroke and 100-200 represents the down stroke.

Posterior Deltoid

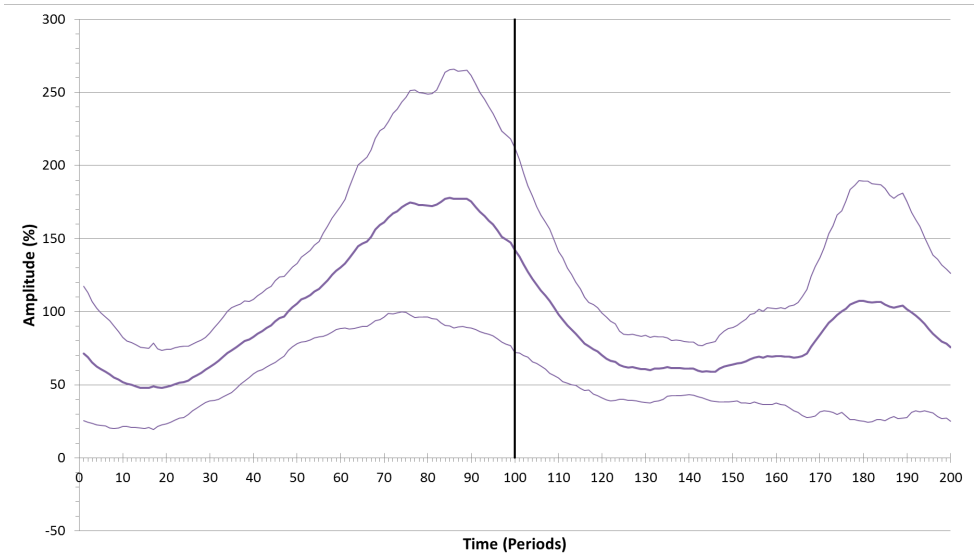

Figure Ap1.5. Graph to show the normal shoulder group (n=19) activation for PD for the movement forward flexion. The thick line and thin line present the mean amplitude and SD(+/-) respectively. The time period 0-100 represent 0 to 180 degrees in the upstroke and 100-200 represents 180-0 degrees in the down stroke.

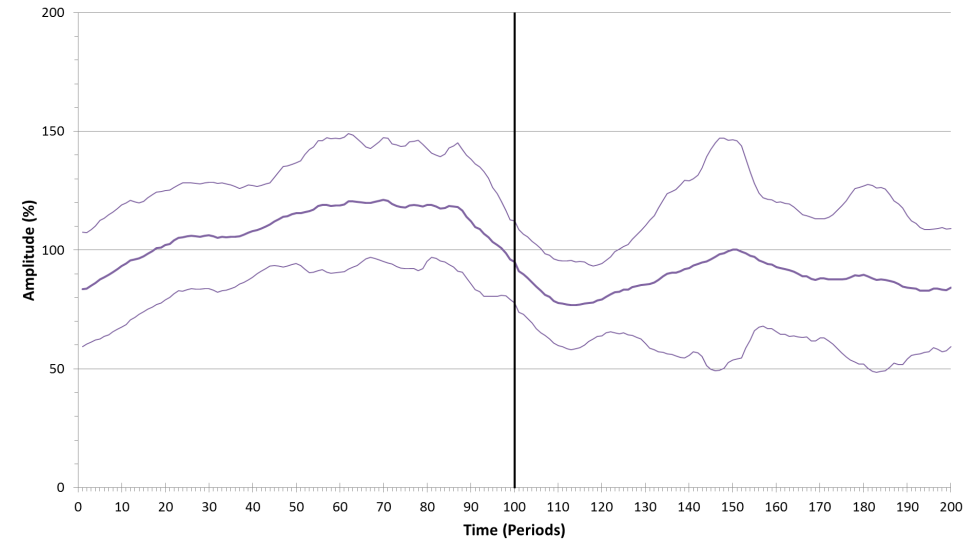

Figure A1.6. Graph to show the normal shoulder group (n=23) activation for PD for the movement forward flexion whilst in the Supine position. The thick line and thin line present the mean amplitude and SD (+/-) respectively. The time period 0-100 represents the upstroke and 100-200 represents the down stroke.

Upper Trapezium

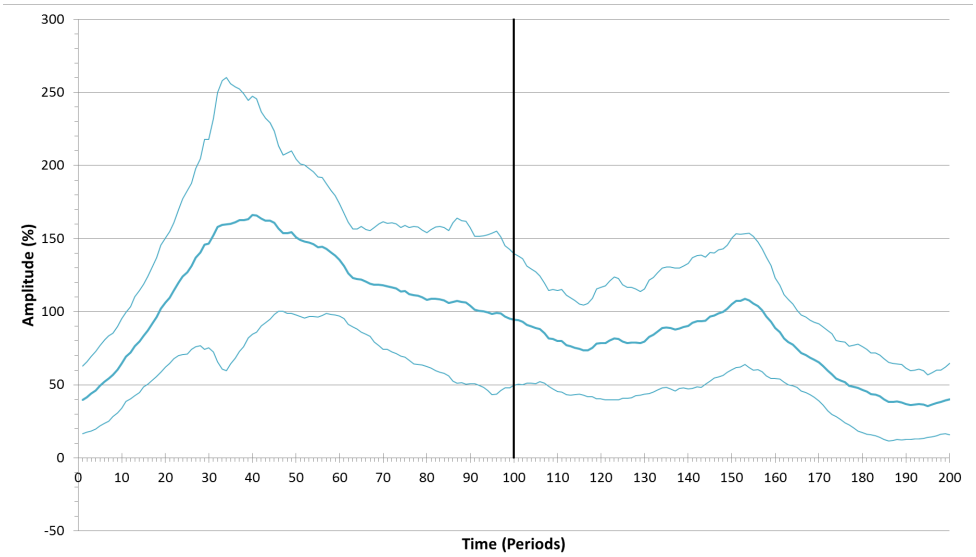

Figure Ap1.7. Graph to show the normal shoulder group (n=17) activation for UT for the movement forward flexion. The thick line and thin line present the mean amplitude and SD (+/-) respectively. The time period 0-100 represent 0 to 180 degrees in the upstroke and 100-200 represents 180-0 degrees in the down stroke.

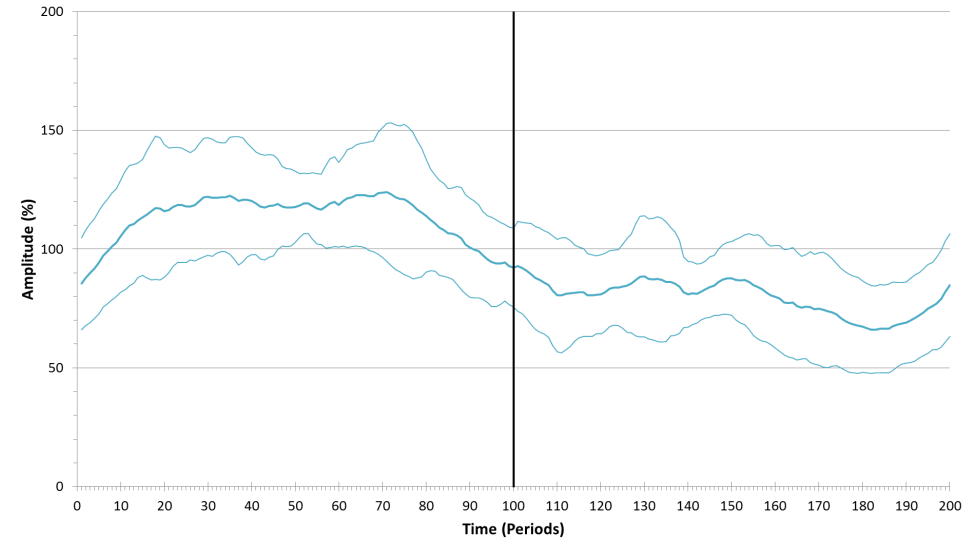

Figure Ap1.8. Graph to show the normal shoulder group (n=16) activation for UT for the movement forward flexion whilst in the Supine position. The thick line and thin line present the mean amplitude and SD (+/-) respectively. The time period 0-100 represents the upstroke and 100-200 represents the down stroke.

Serratus Anterior

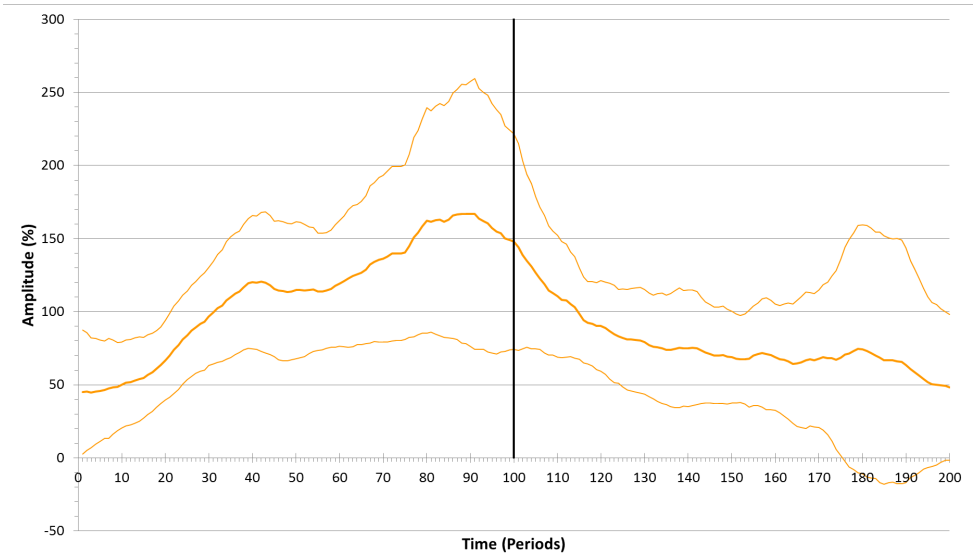

Figure A1.9. Graph to show the normal shoulder group (n=16) activation for AD for the movement forward flexion. The thick line and thin line present the mean amplitude and SD (+/-) respectively. The time period 0-100 represent 0 to 180 degrees in the upstroke and 100-200 represents 180-0 degrees in the down stroke.

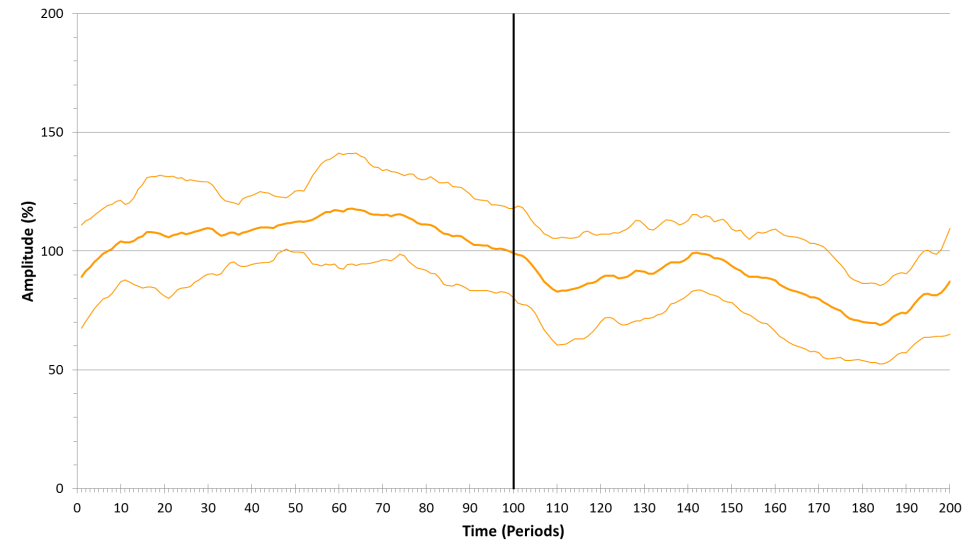

Figure A1.10. Graph to show the normal shoulder group (n=16) activation for SA for the movement forward flexion whilst in the Supine position. The thick line and thin line present the mean amplitude and SD (+/-) respectively. The time period 0-100 represents the upstroke and 100-200 represents the down stroke.

Teres Major

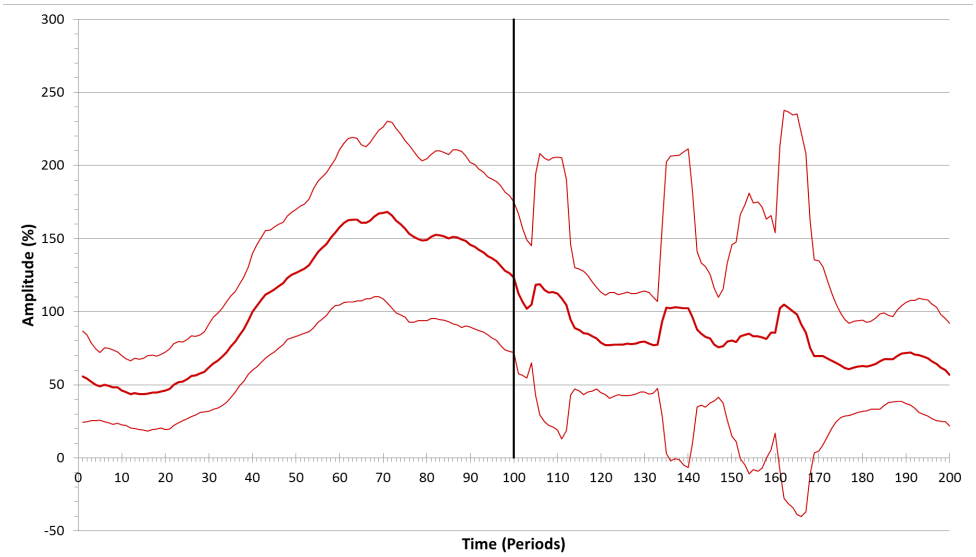

Figure A1.11. Graph to show the normal shoulder group (n=13) activation for TM for the movement forward flexion. The thick line and thin line present the mean amplitude and SD (+/-) respectively. The time period 0-100 represent 0 to 180 degrees in the upstroke and 100-200 represents 180-0 degrees in the down stroke. Note the periods of very high variation in Phase 2 (see text).

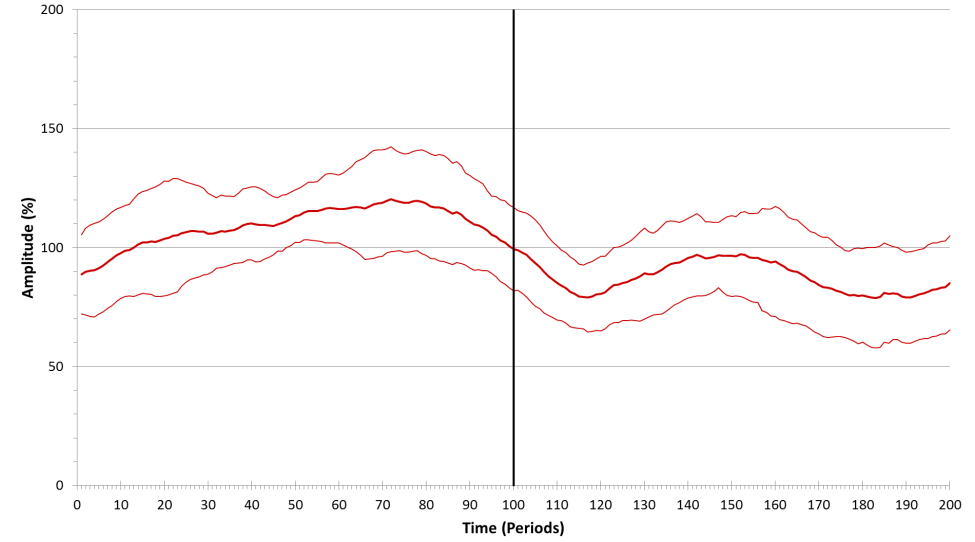

Figure A1.12. Graph to show the normal shoulder group (n=19) activation for TM for the movement forward flexion whilst in the Supine position. The thick line and thin line present the mean amplitude and SD (+/-) respectively. The time period 0-100 represents the upstroke and 100-200 represents the down stroke.

Latissimus Dorsi

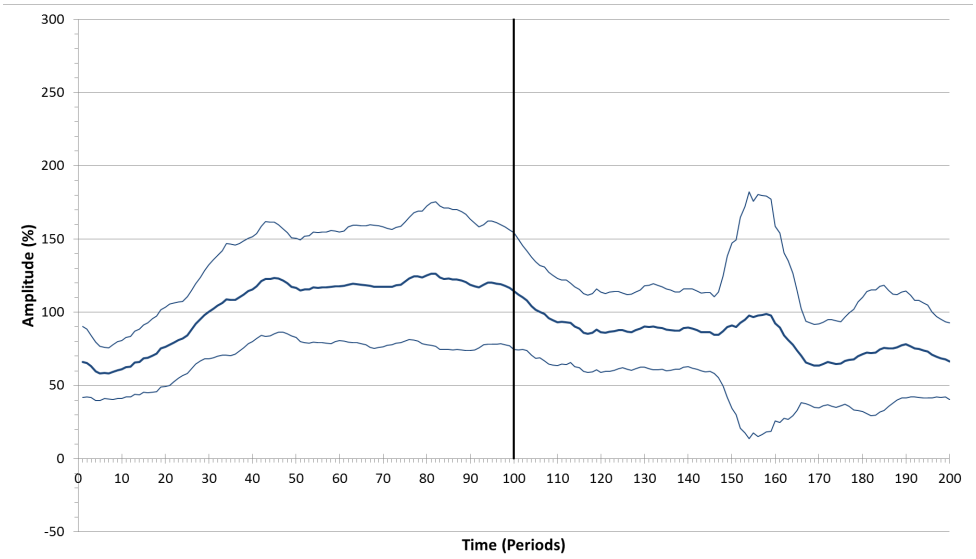

Figure Ap13. Graph to show the normal shoulder group (n=17) activation for LD for the movement forward flexion. The thick line and thin line present the mean amplitude and SD (+/-) respectively. The time period 0-100 represent 0 to 180 degrees in the upstroke and 100-200 represents 180-0 degrees in the down stroke.

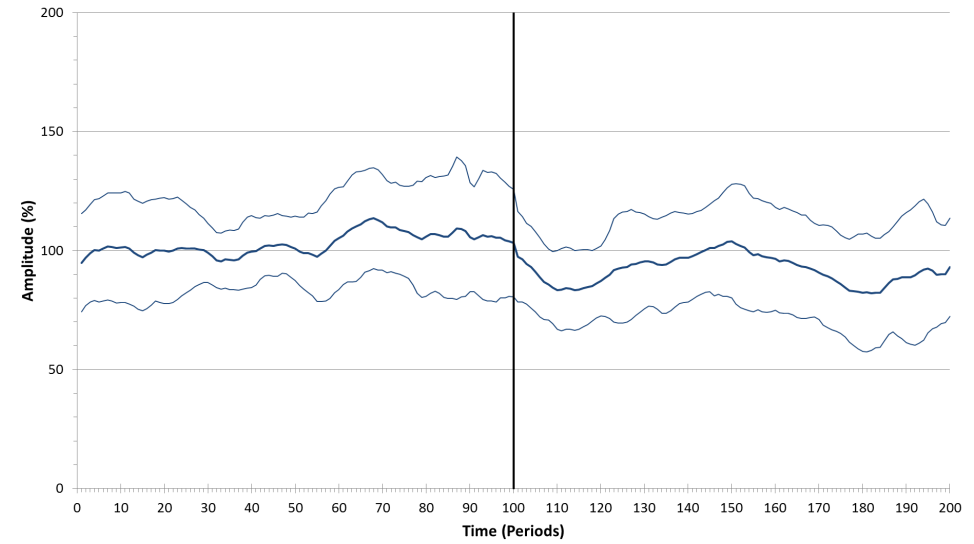

Figure A1.14. Graph to show the normal shoulder group (n=22) activation for LD for the movement forward flexion whilst in the Supine position. The thick line and thin line present the mean amplitude and SD (+/-) respectively. The time period 0-100 represents the upstroke and 100-200 represents the down stroke.

Pectoralis Major

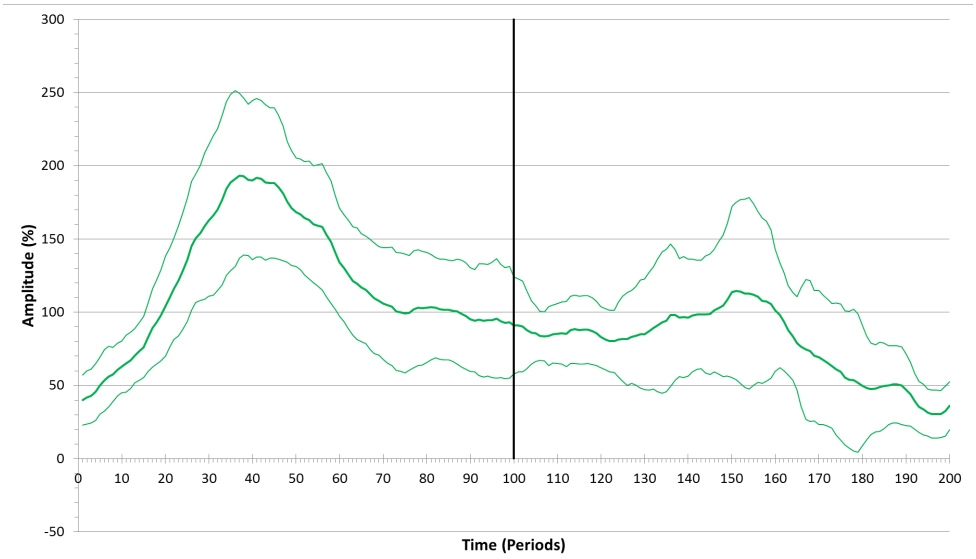

Figure A1.15. Graph to show the normal shoulder group (n=19) activation for PM for the movement forward flexion. The thick line and thin line present the mean amplitude and SD (+/-) respectively. The time period 0-100 represent 0 to 180 degrees in the upstroke and 100-200 represents 180-0 degrees in the down stroke.

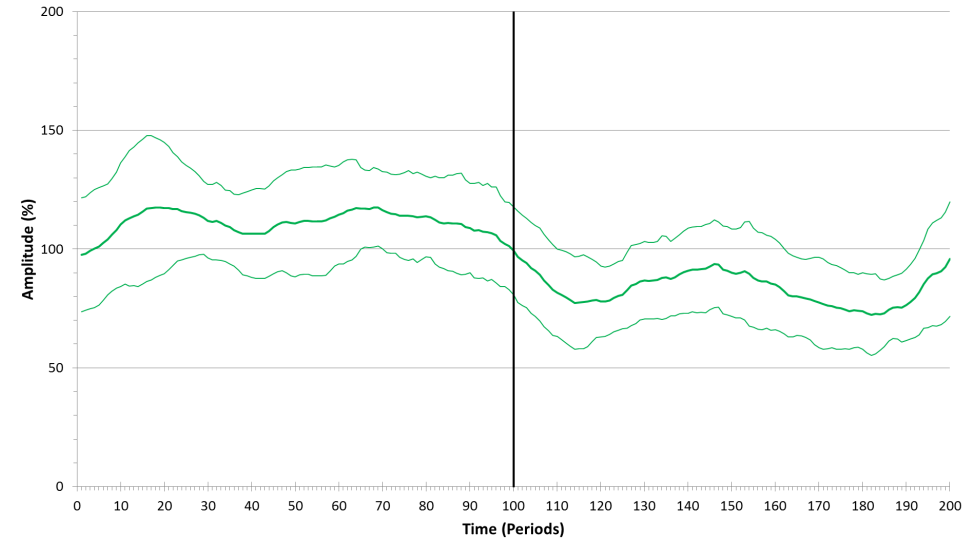

Figure A1.16. Graph to show the normal shoulder group (n=23) activation for PM for the movement forward flexion whilst in the Supine position. The thick line and thin line present the mean amplitude and SD (+/-) respectively. The time period 0-100 represents the upstroke and 100-200 represents the down stroke.

Biceps Brachii

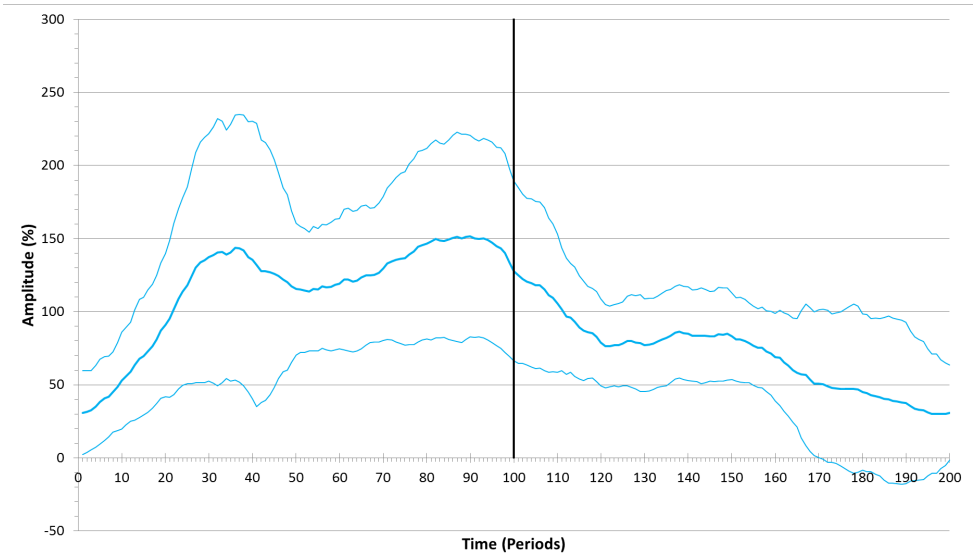

Figure A1.17. Graph to show the normal shoulder group (n=19) activation for BB for the movement forward flexion. The thick line and thin line present the mean amplitude and SD (+/-) respectively. The time period 0-100 represent 0 to 180 degrees in the upstroke and 100-200 represents 180-0 degrees in the down stroke.

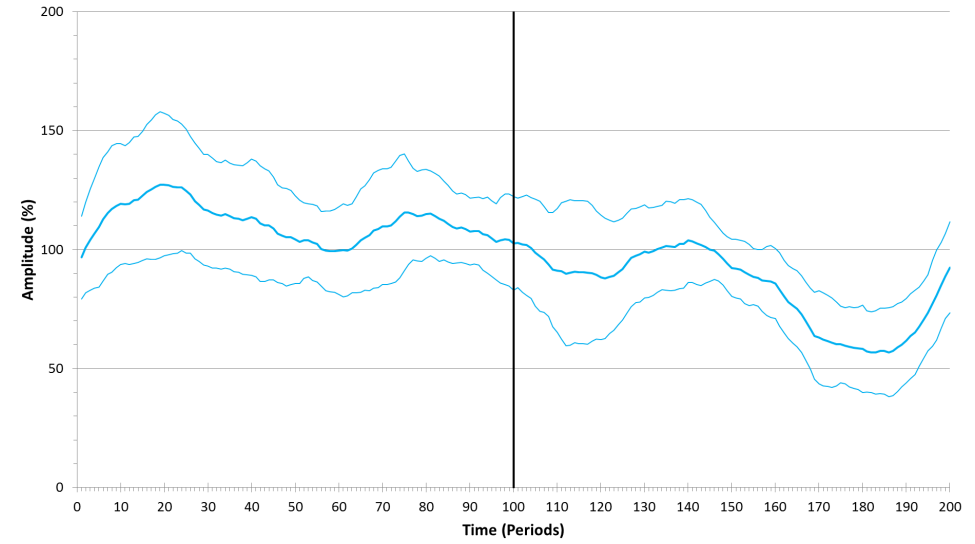

Figure A1.18. Graph to show the normal shoulder group (n=22) activation for AD for the movement forward flexion whilst in the Supine position. The thick line and thin line present the mean amplitude and SD (+/-) respectively. The time period 0-100 represents the upstroke and 100-200 represents the down stroke.

Supraspinatus

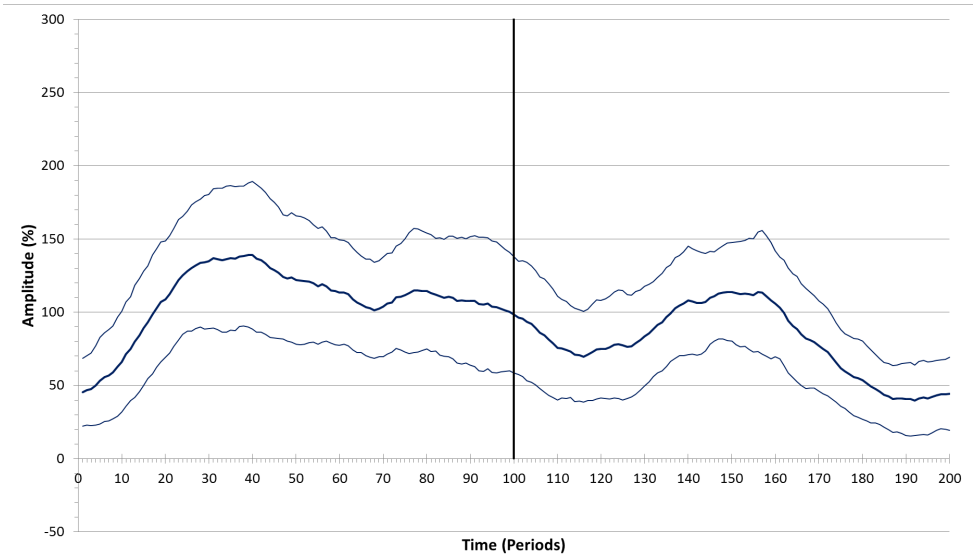

Figure A.19. Graph to show the normal shoulder group (n=19) activation for SSP for the movement forward flexion. The thick line and thin line present the mean amplitude and SD (+/-) respectively. The time period 0-100 represent 0 to 180 degrees in the upstroke and 100-200 represents 180-0 degrees in the down stroke.

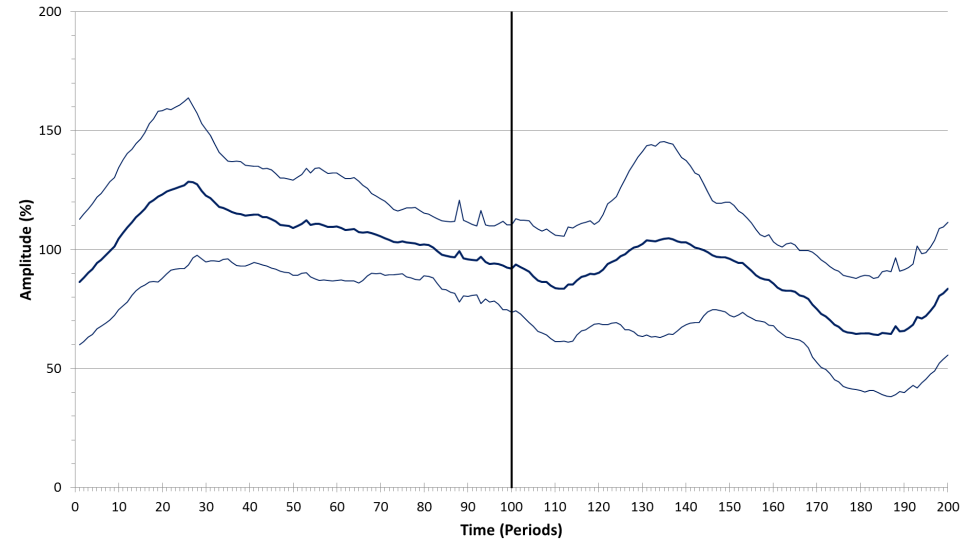

Figure A1.20. Graph to show the normal shoulder group (n=21) activation for SSP for the movement forward flexion whilst in the Supine position. The thick line and thin line present the mean amplitude and SD (+/-) respectively. The time period 0-100 represents the upstroke and 100-200 represents the down stroke.

Infraspinatus

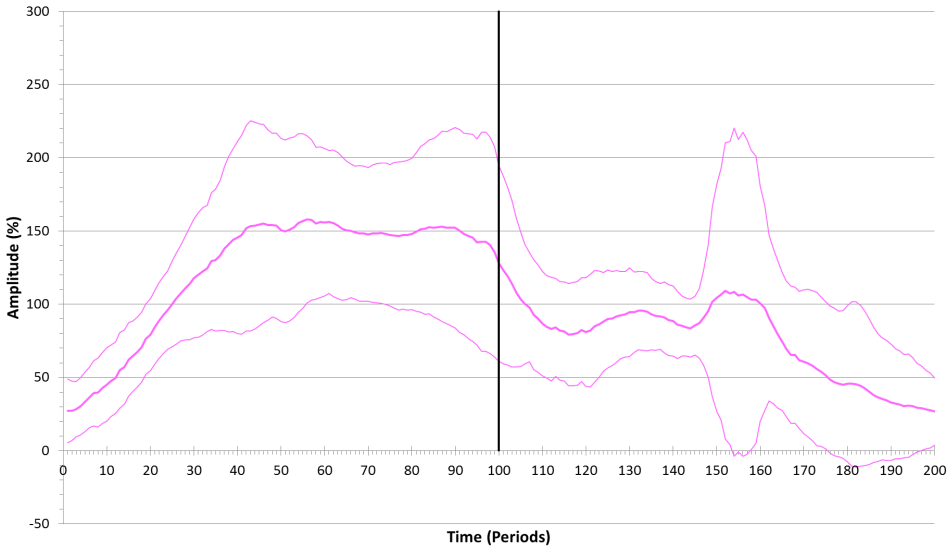

Figure A1.21. Graph to show the normal shoulder group (n=19) activation for SSP for the movement forward flexion. The thick line and thin line present the mean amplitude and SD (+/-) respectively. The time period 0-100 represent 0 to 180 degrees in the upstroke and 100-200 represents 180-0 degrees in the down stroke.

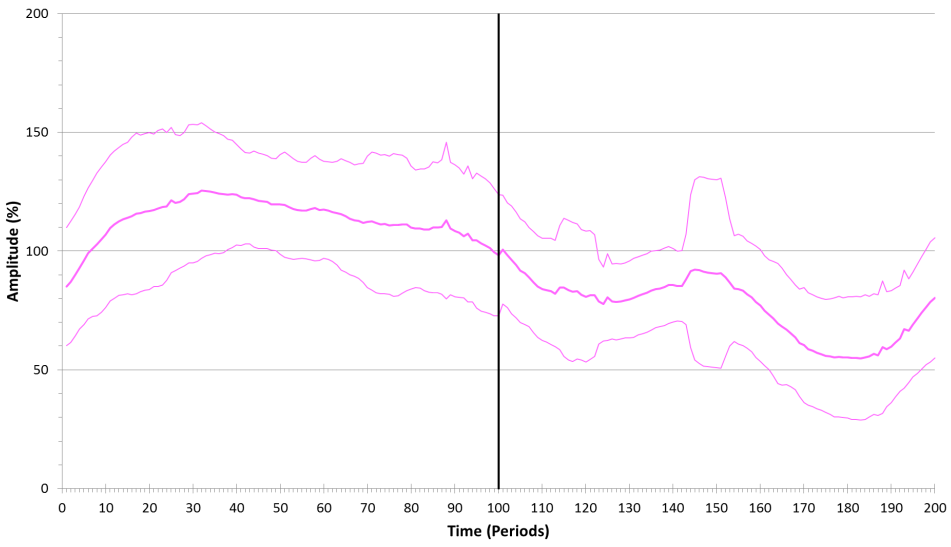

Figure A1.22. Graph to show the normal shoulder group (n=21) activation for ISP for the movement forward flexion whilst in the Supine position. The thick line and thin line present the mean amplitude and SD (+/-) respectively. The time period 0-100 represents the upstroke and 100-200 represents the down stroke.

Subscapularis

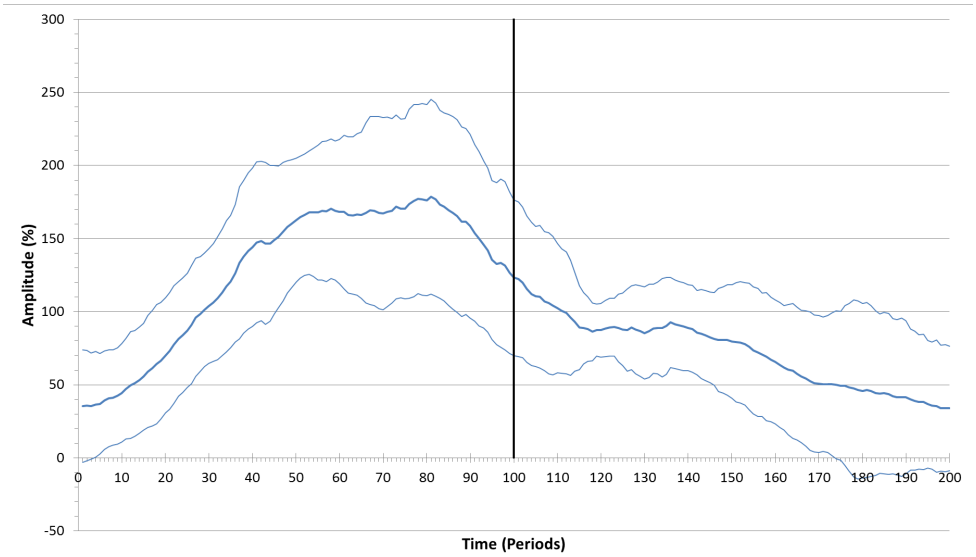

Figure A1.23. Graph to show the normal shoulder group (n=19) activation for SUB for the movement forward flexion. The thick line and thin line present the mean amplitude and SD (+/-) respectively. The time period 0-100 represent 0 to 180 degrees in the upstroke and 100-200 represents 180-0 degrees in the down stroke.

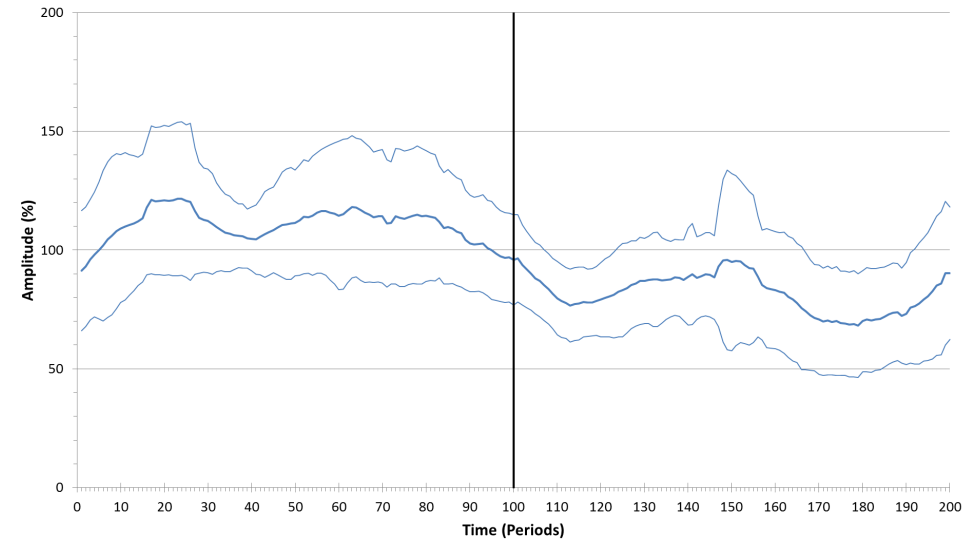

Figure A1.24. Graph to show the normal shoulder group (n=14) activation for SUB for the movement forward flexion whilst in the Supine position. The thick line and thin line present the mean amplitude and SD (+/-) respectively. The time period 0-100 represents the upstroke and 100-200 represents the down stroke.

Appendix 2 – EMG – Comparative Study – Forward Flexio

Anterior Deltoid

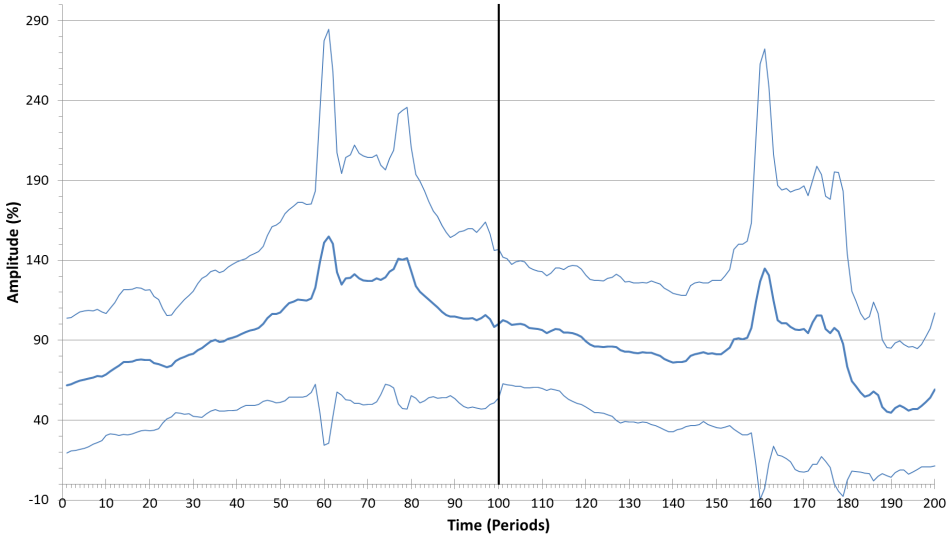

Figure 2.1. Graph to show the Patient group (n=14) activation for AD for the movement forward flexion. The thick line and thin line present the mean amplitude and SD(+/-) respectively. The time period 0-100 represent 0 to 90 degrees in the upstroke and 100-200 represents 90-0 degrees in the down stroke.

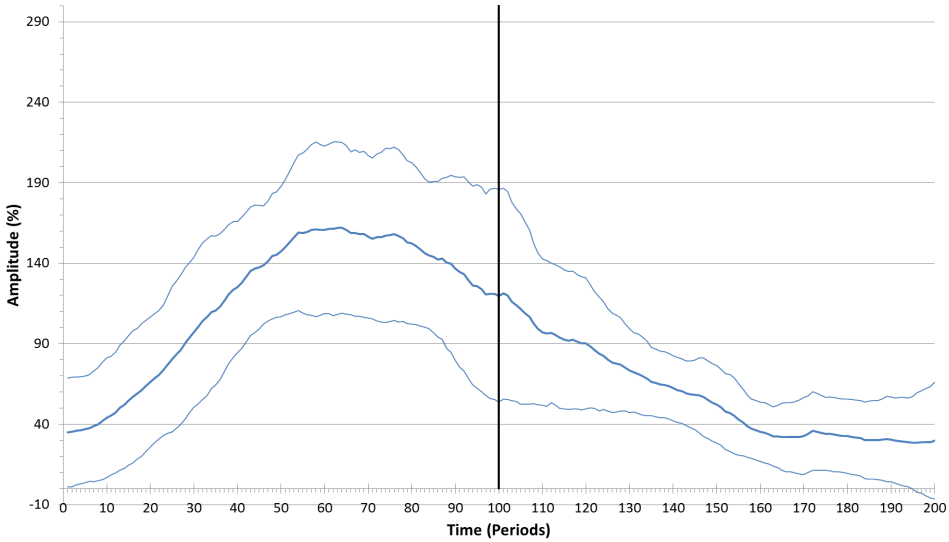

Figure 2.2. Graph to show the Control group (n=10) activation for AD for the movement forward flexion. The thick line and thin line present the mean amplitude and SD (+/-) respectively. The time period 0-100 represent 0 to 90 degrees in the upstroke and 100-200 represent 90-0 degrees in the down stroke.

7.1.1.1 Middle Deltoid

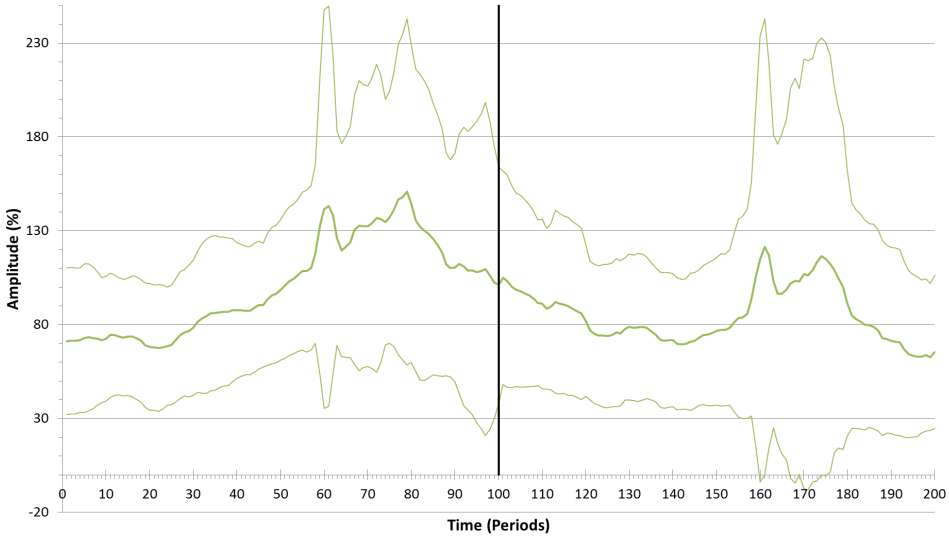

Figure 2.3. Graph to show the Patient group (n=14) activation for MD for the movement forward flexion. The thick line and thin line present the mean amplitude and SD (+/-) respectively. The time period 0-100 represent 0 to 90 degrees in the upstroke and 100-200 represents 90-0 degrees in the down stroke.

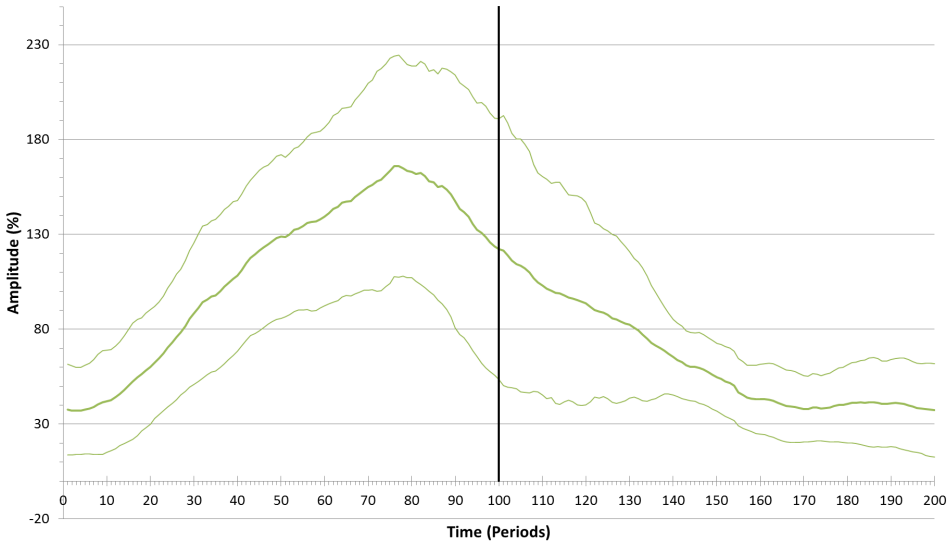

Figure 2.4. Graph to show the Control group (n=12) activation for MD for the movement forward flexion. The thick line and thin line present the mean amplitude and SD (+/-) respectively. The time period 0-100 represent 0 to 90 degrees in the upstroke and 100-200 represents 90-0 degrees in the down stroke.

Posterior Deltoid

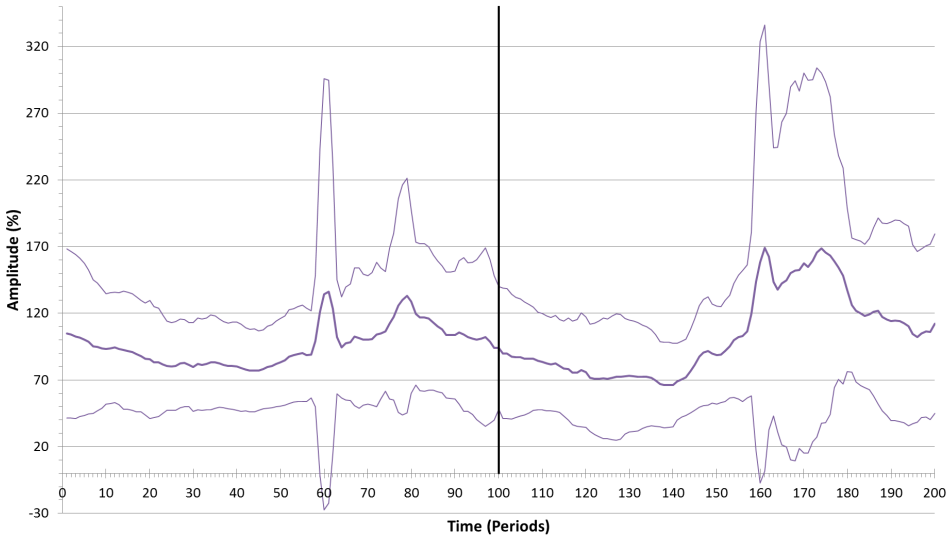

Figure 2.5. Graph to show the Patient group (n=14) activation for PD for the movement forward flexion. The thick line and thin line present the mean amplitude and SD(+/-) respectively. The time period 0-100 represent 0 to 90 degrees in the upstroke and 100-200 represents 90-0 degrees in the down stroke.

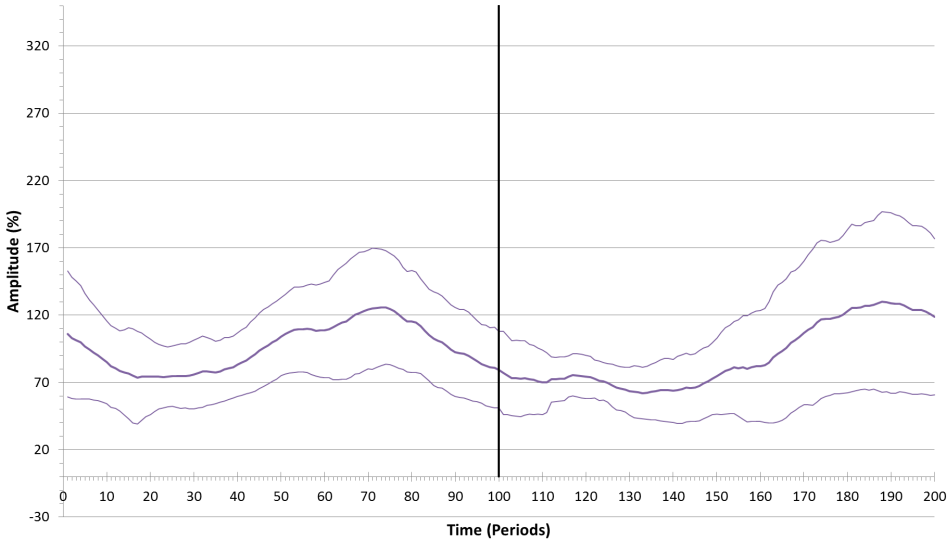

Figure 2.6. Graph to show the Control group (n=9) activation for PD for the movement forward flexion. The thick line and thin line present the mean amplitude and SD (+/-) respectively. The time period 0-100 represent 0 to 90 degrees in the upstroke and 100-200 represent 90-0 degrees in the down stroke.

Upper Trapezium

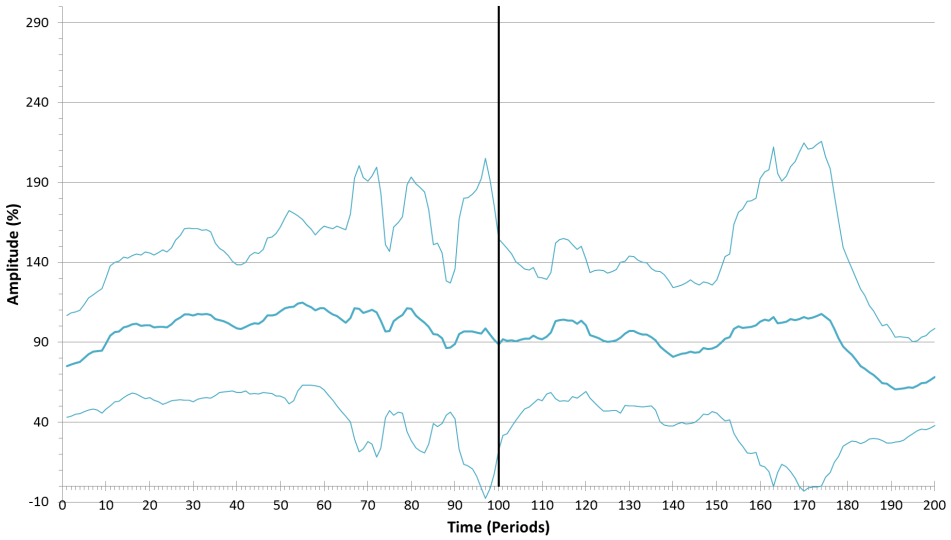

Figure A2.7. Graph to show the Patient group (n=14) activation for UT for the movement forward flexion. The thick line and thin line present the mean amplitude and SD (+/-) respectively. The time period 0-100 represent 0 to 90 degrees in the upstroke and 100-200 represent 90-0 degrees in the down stroke.

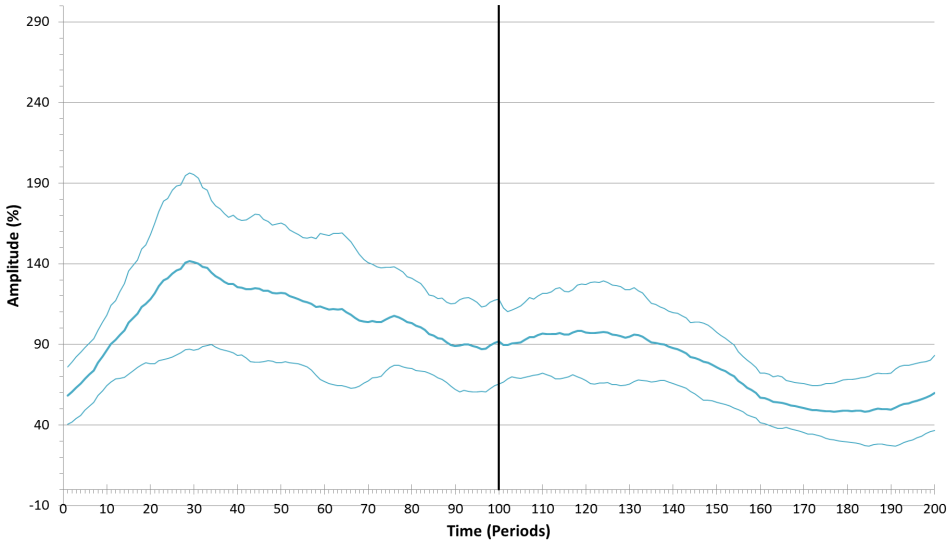

Figure A2.8. Graph to show the Control group (n=10) activation for UT for the movement forward flexion. The thick line and thin line present the mean amplitude and SD (+/-) respectively. The time period 0-100 represent 0 to 90 degrees in the upstroke and 100-200 represents 90-0 degrees in the down stroke.

Serratus Anterior

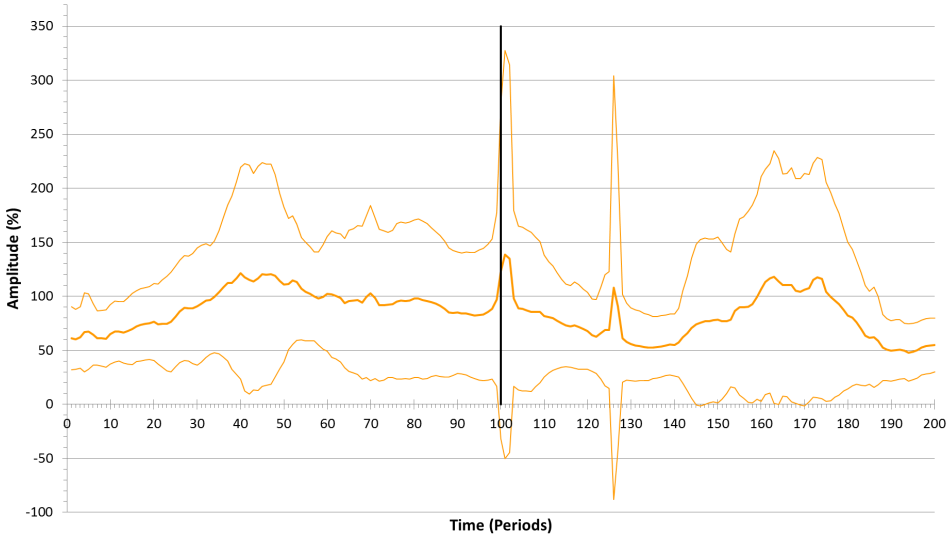

Figure A2.9. Graph to show the Patient group (n=14) activation for SA for the movement forward flexion. The thick line and thin line present the mean amplitude and SD (+/-) respectively. The time period 0-100 represent 0 to 90 degrees in the upstroke and 100-200 represents 90-0 degrees in the down stroke.

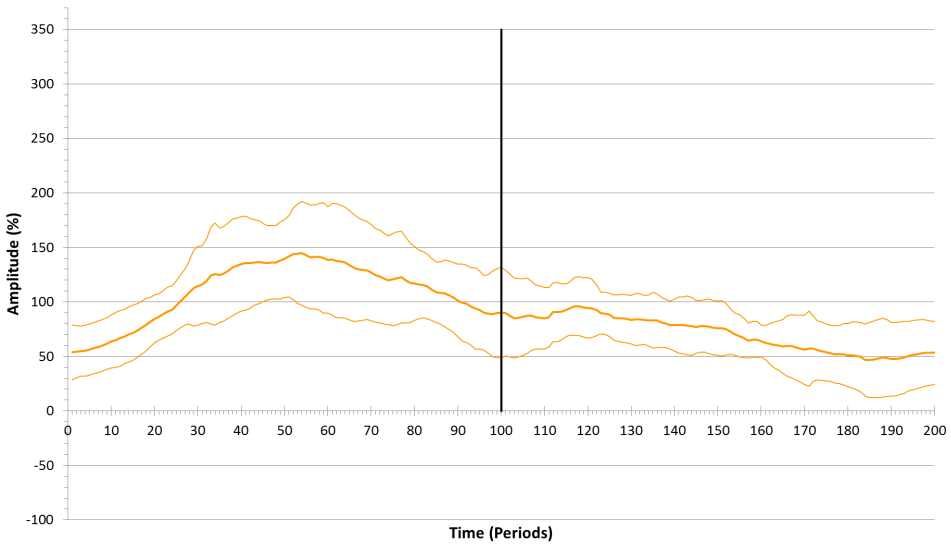

Figure A2.10. Graph to show the Control group (n=9) activation for SA for the movement forward flexion. The thick line and thin line present the mean amplitude and SD (+/-) respectively. The time period 0-100 represent 0 to 90 degrees in the upstroke and 100-200 represent 90-0 degrees in the down stroke.

Teres Major

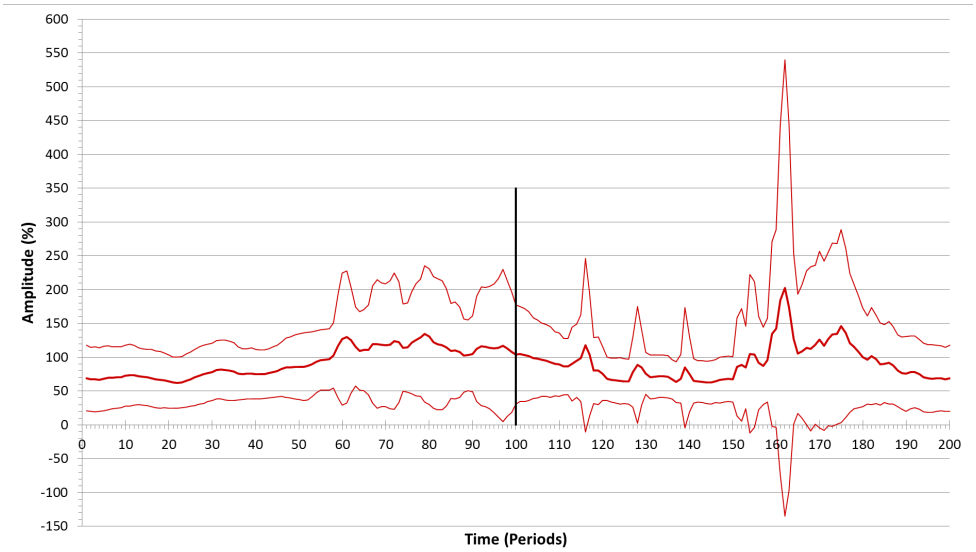

Figure A2.11. Graph to show the Patient group (n=14) activation for TM for the movement forward flexion. The thick line and thin line present the mean amplitude and SD (+/-) respectively. The time period 0-100 represent 0 to 90 degrees in the upstroke and 100-200 represents 90-0 degrees in the down stroke.

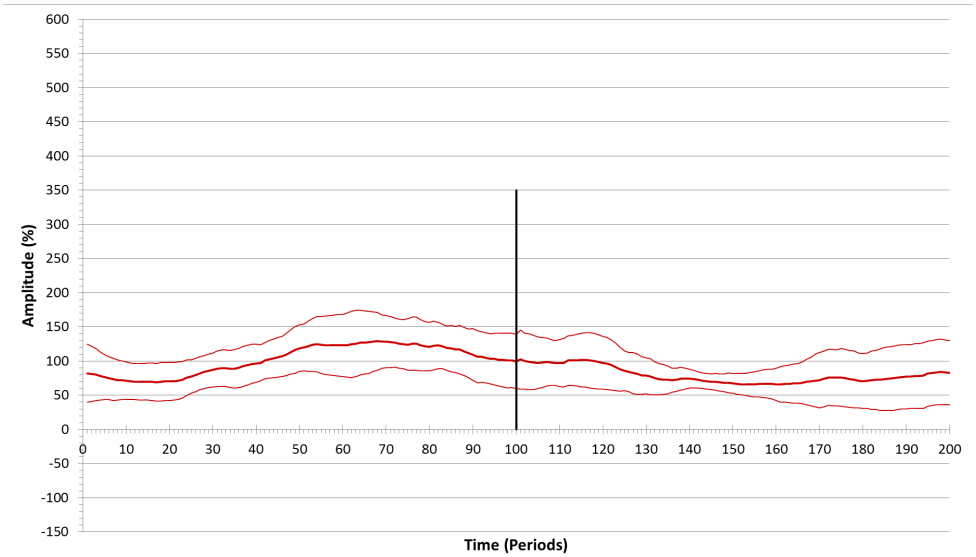

Figure A1.12. Graph to show the Control group (n=11) activation for TM for the movement forward flexion. The thick line and thin line present the mean amplitude and SD (+/-) respectively. The time period 0-100 represent 0 to 90 degrees in the upstroke and 100-200 represent 90-0 degrees in the down stroke.

Latissimus Dorsi

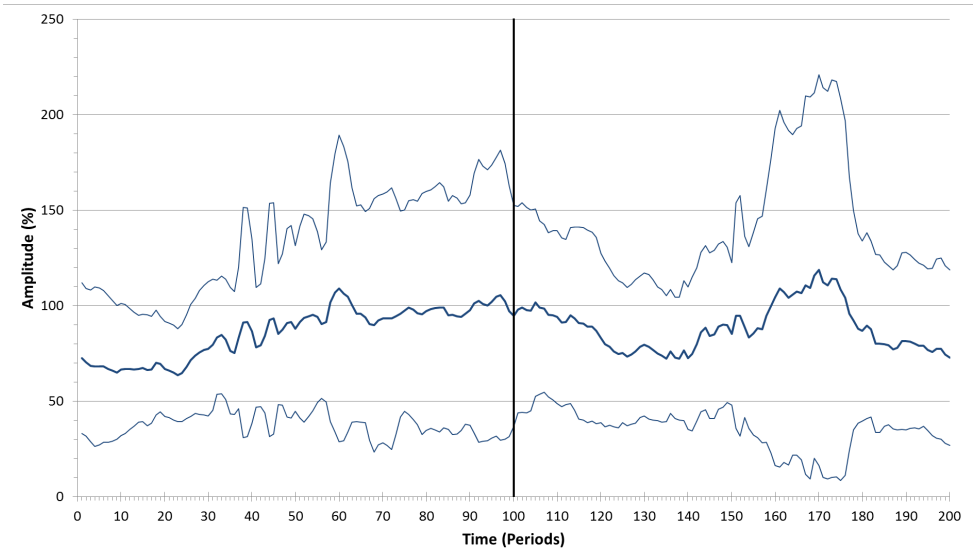

Figure A2.13. Graph to show the Patient group (n=14) activation for LD for the movement forward flexion. The thick line and thin line present the mean amplitude and SD (+/-) respectively. The time period 0-100 represent 0 to 90 degrees in the upstroke and 100-200 represents 90-0 degrees in the down stroke.

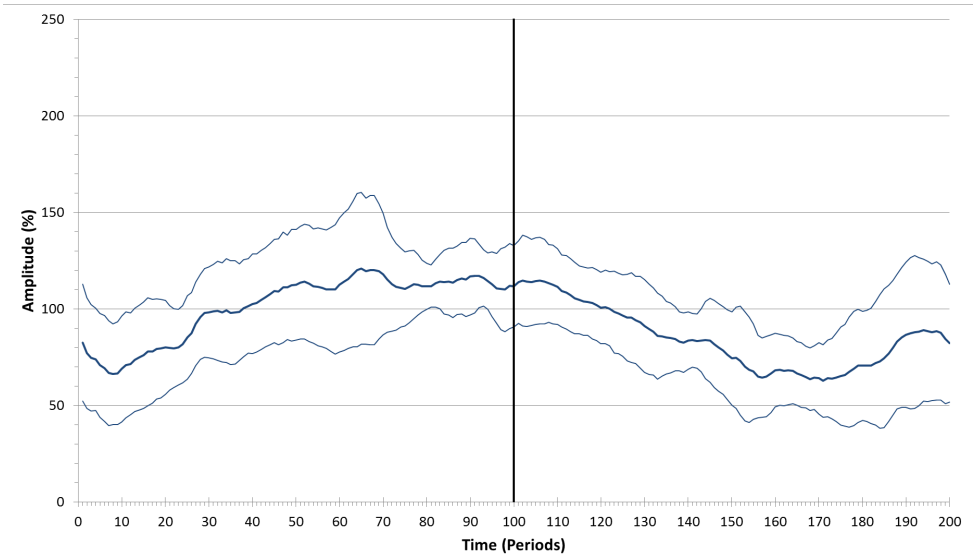

Figure A2.14. Graph to show the Control group (n=9) activation for LD for the movement forward flexion. The thick line and thin line present the mean amplitude and SD (+/-) respectively. The time period 0-100 represent 0 to 90 degrees in the upstroke and 100-200 represents 90-0 degrees in the down stroke.

Pectoralis Major

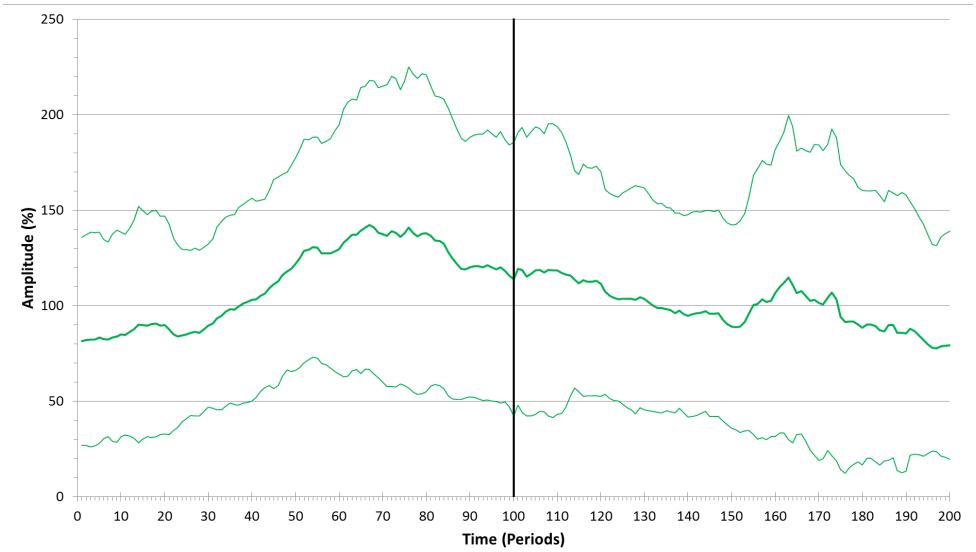

Figure A2.15. Graph to show the Patient group (n=14) activation for PM for the movement forward flexion. The thick line and thin line present the mean amplitude and SD (+/-) respectively. The time period 0-100 represent 0 to 90 degrees in the upstroke and 100-200 represent 90-0 degrees in the down stroke.

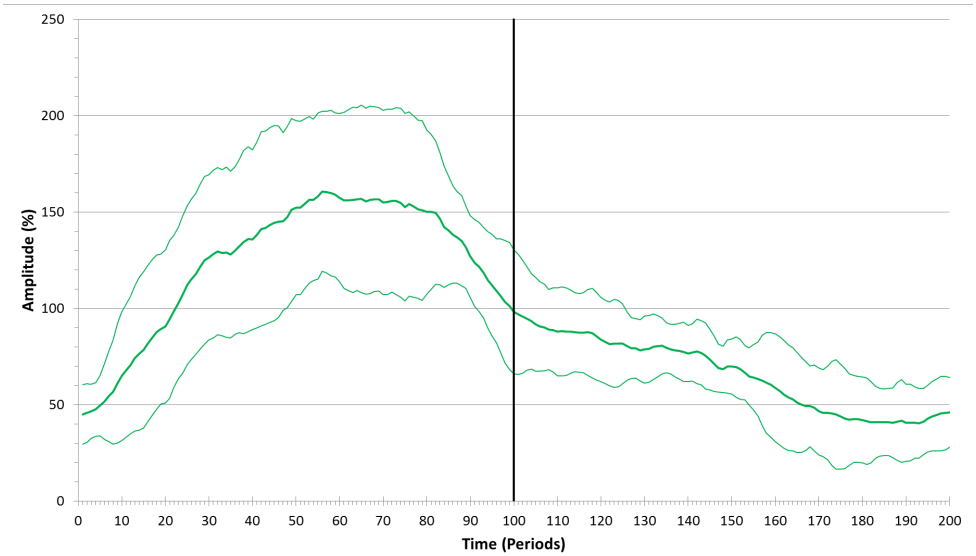

Figure A2.16. Graph to show the Control group (n=8) activation for PM for the movement forward flexion. The thick line and thin line present the mean amplitude and SD (+/-) respectively. The time period 0-100 represent 0 to 90 degrees in the upstroke and 100-200 represent 90-0 degrees in the down stroke.

Biceps Brachii

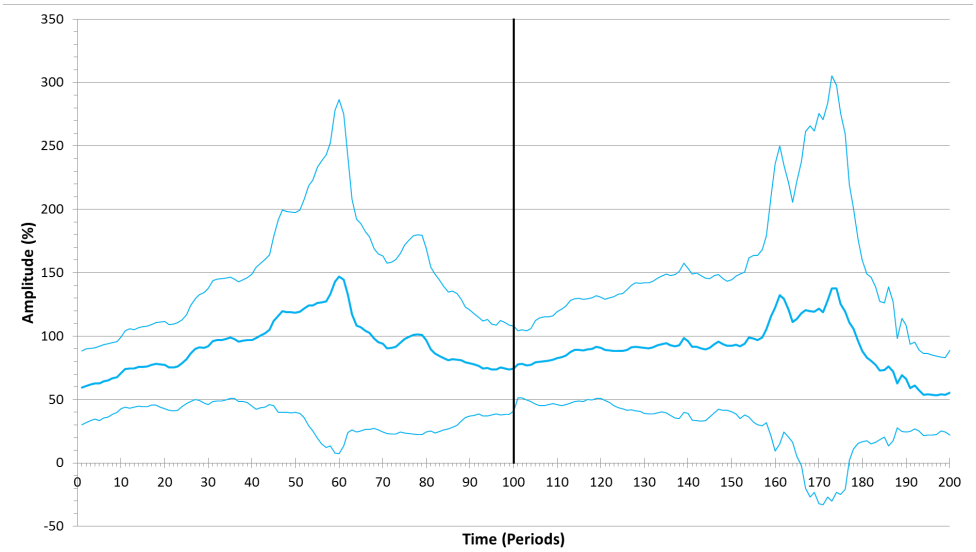

Figure A2.17. Graph to show the Patient group (n=14) activation for BB for the movement forward flexion. The thick line and thin line present the mean amplitude and SD (+/-) respectively. The time period 0-100 represent 0 to 90 degrees in the upstroke and 100-200 represents 90-0 degrees in the down stroke.

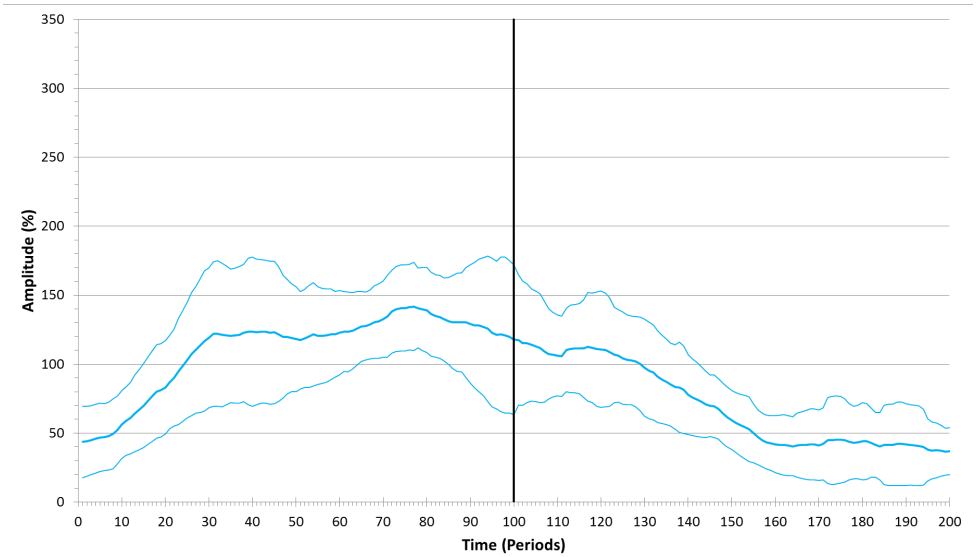

Figure A2.18. Graph to show the Control group (n=11) activation for BB for the movement forward flexion. The thick line and thin line present the mean amplitude and SD (+/-) respectively. The time period 0-100 represent 0 to 90 degrees in the upstroke and 100-200 represent 90-0 degrees in the down stroke.

Infraspinatus

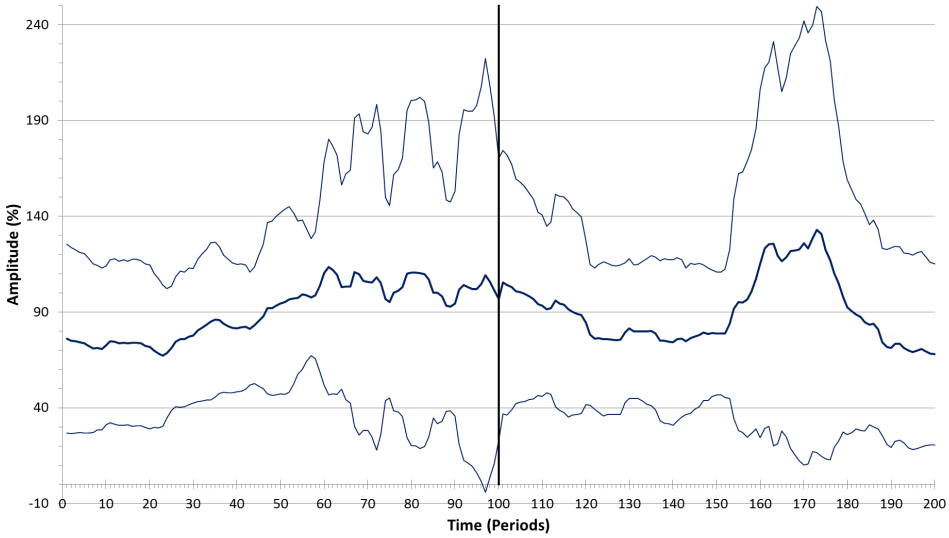

Figure A1.9. Graph to show the Patient group (n=14) activation for ISP for the movement forward flexion. The thick line and thin line present the mean amplitude and SD (+/-) respectively. The time period 0-100 represent 0 to 90 degrees in the upstroke and 100-200 represents 90-0 degrees in the down stroke.

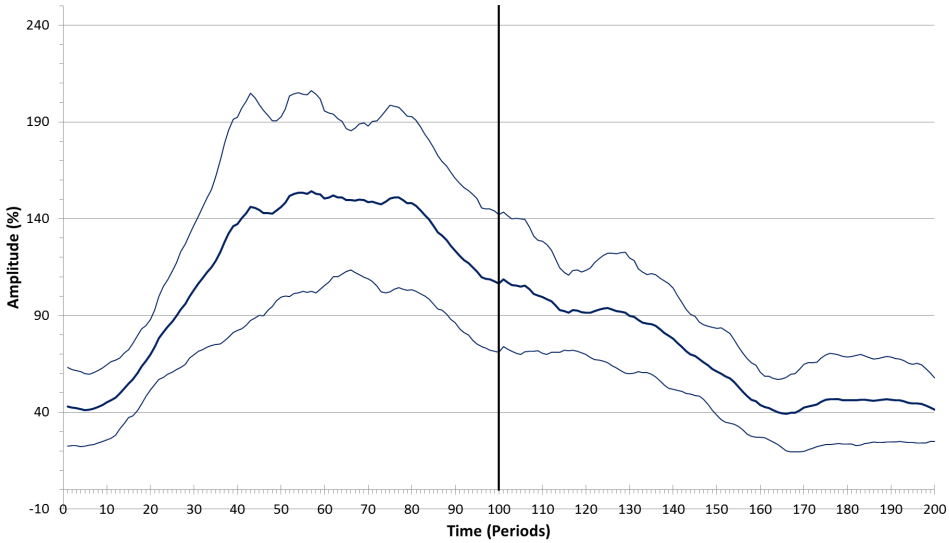

Figure A2.20. Graph to show the Control group (n=10) activation for ISP for the movement forward flexion. The thick line and thin line present the mean amplitude and SD (+/-) respectively. The time period 0-100 represent 0 to 90 degrees in the upstroke and 100-200 represents 90-0 degrees in the down stroke.

Appendix 3 – EMG – Normal Shoulder Group - Abduction

Anterior Deltoid

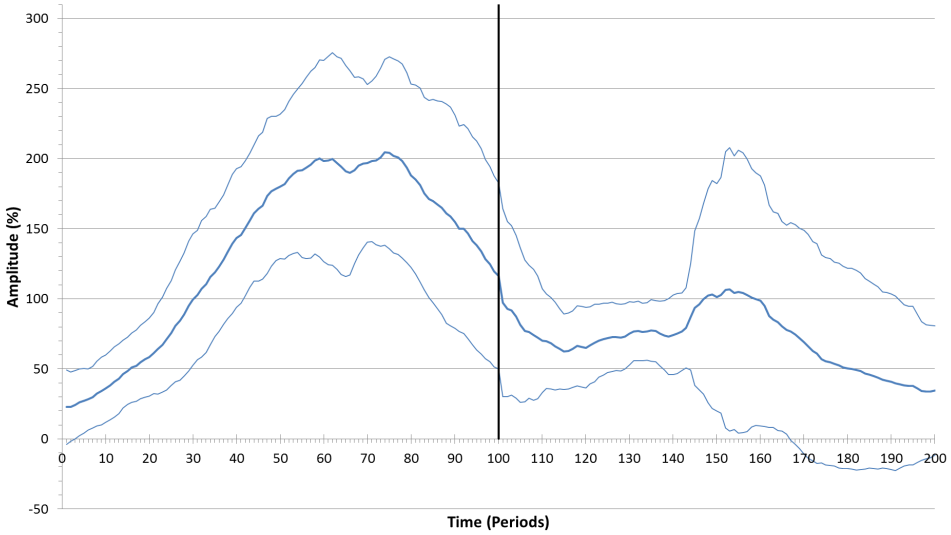

Figure A3.1. Graph to show the normal shoulder group (n=19) activation for AD for the movement abduction. The thick line and thin line present the mean amplitude and SD (+/-) respectively. The time period 0-100 represent 0 to 180 degrees in the upstroke and the time period 100-200 represent 180-0 degrees in the down stroke.

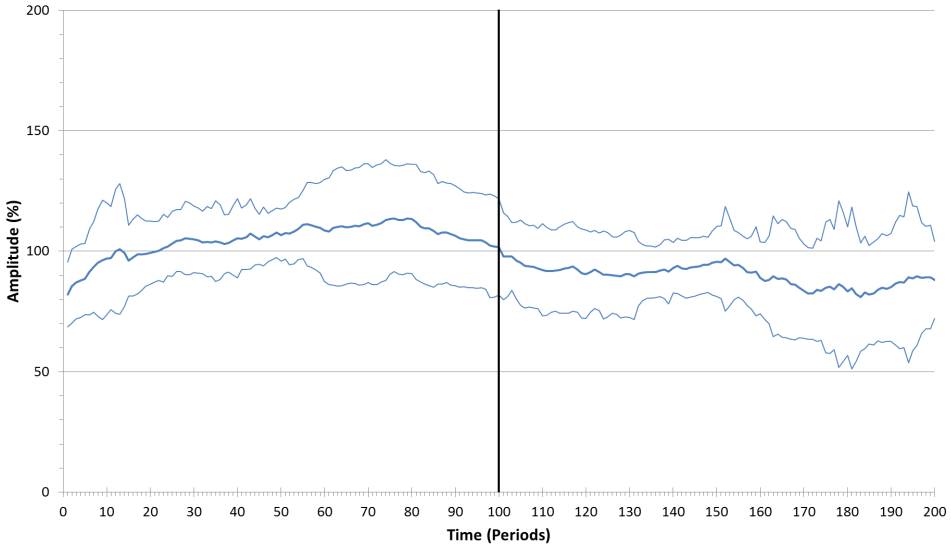

Figure A3.2. Graph to show the normal shoulder group (n=20) activation for AD for the movement abduction/adduction whilst in the Supine position. The thick line and thin line present the mean amplitude and SD (+/-) respectively. The time period 0-100 represents the upstroke and the time period 100-200 represents the down stroke.

Middle Deltoid

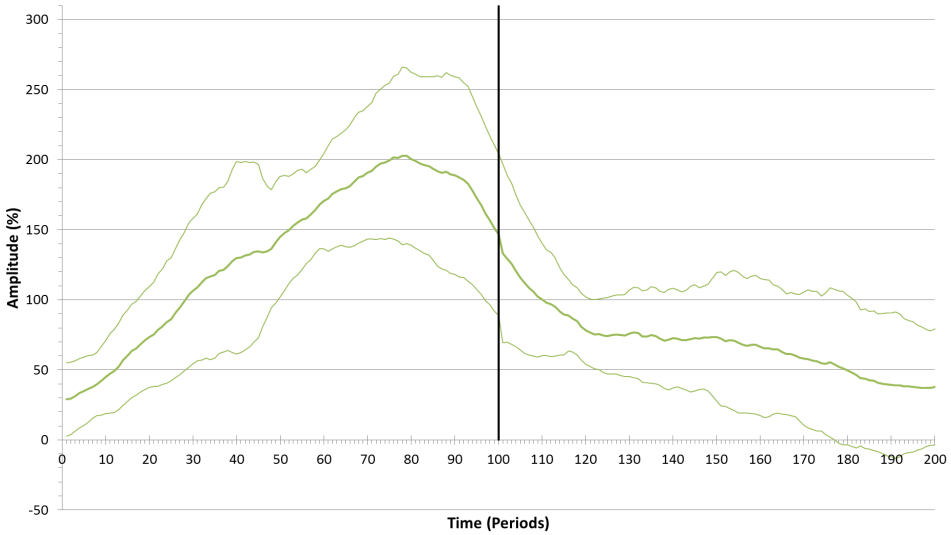

Figure A3.4. Graph to show the normal shoulder group (n=18) activation for MD for the movement abduction. The thick line and thin line present the mean amplitude and SD (+/-) respectively. The time period 0-100 represent 0 to 180 degrees in the upstroke and 100-200 represents 180-0 degrees in the down stroke.

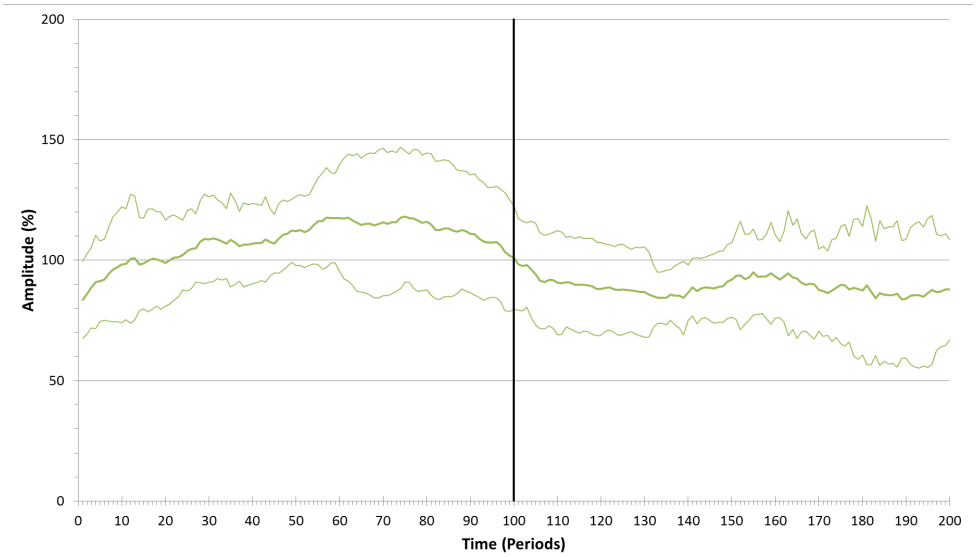

Figure A3.5. Graph to show the normal shoulder group (n=18) activation for MD for the movement abduction/addution whilst in the Supine position. The thick line and thin line present the mean amplitude and SD (+/-) respectively. The time period 0-100 represents the upstroke and 100-200 represents the down stroke.

Posterior Deltoid

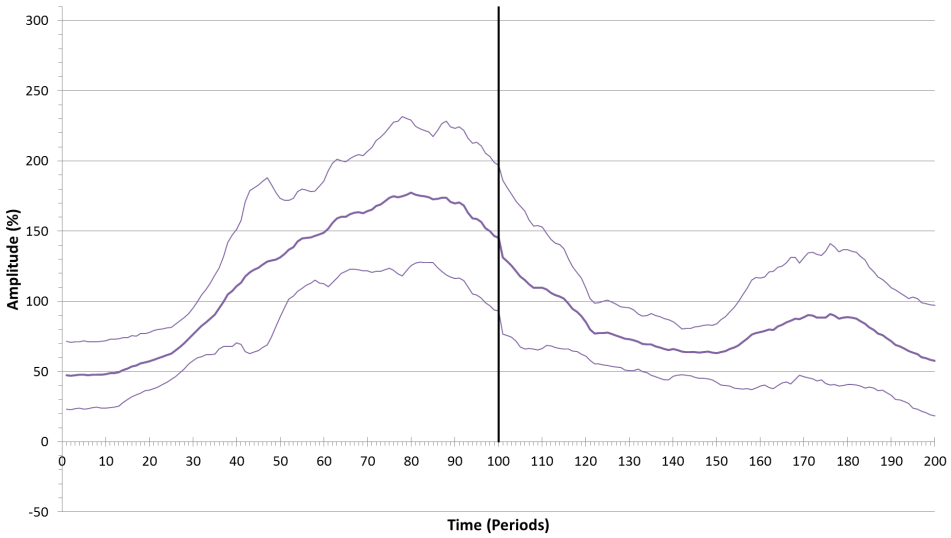

Figure A3.6. Graph to show the normal shoulder group (n=18) activation for PD for the movement abduction. The thick line and thin line present the mean amplitude and SD (+/-) respectively. The time period 0-100 represent 0 to 180 degrees in the upstroke and 100-200 represents 180-0 degrees in the down stroke.

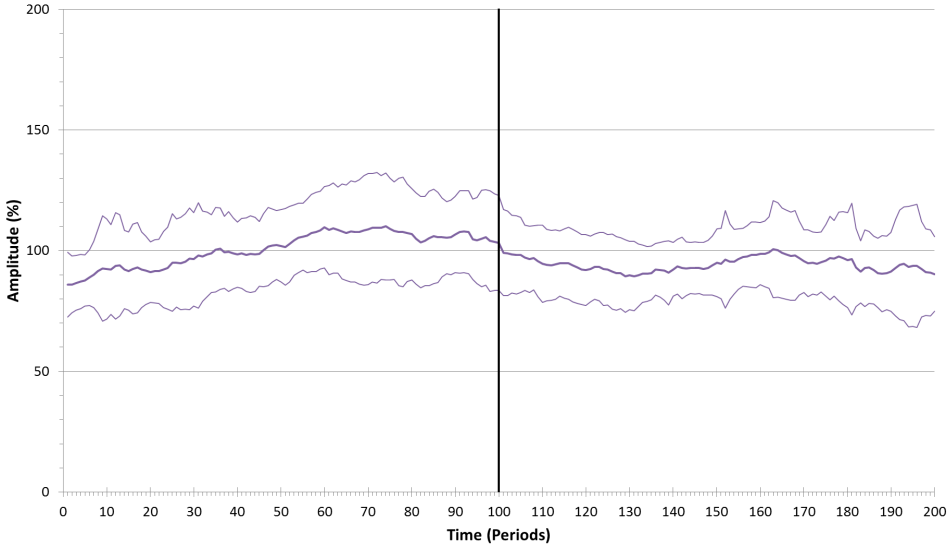

Figure A3.7. Graph to show the normal shoulder group (n=17) activation for PD for the movement abduction/adduction whilst in the Supine position. The thick line and thin line present the mean amplitude and SD (+/-) respectively. The time period 0-100 represents the upstroke and 100-200 represents the down stroke.

Upper Trapezium

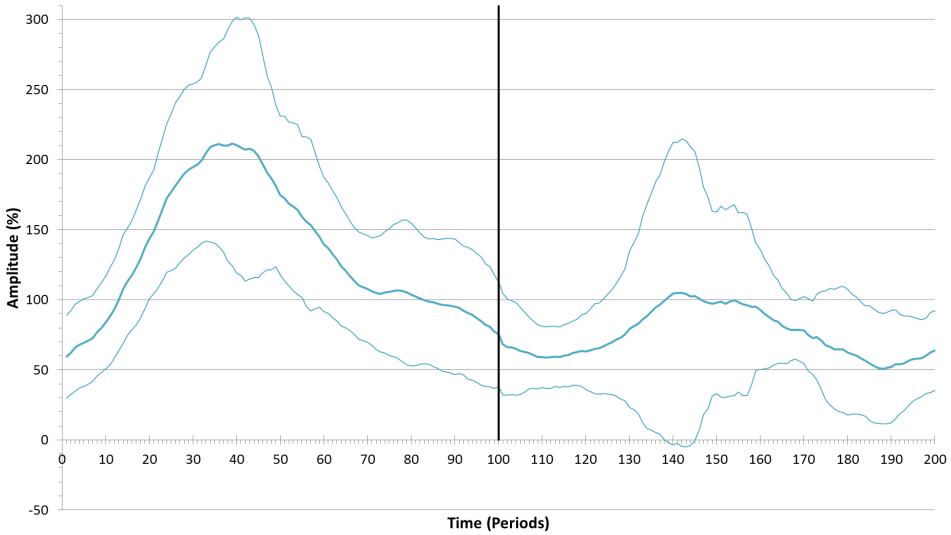

Figure A3.8. Graph to show the normal shoulder group (n=17) activation for UT for the movement abduction. The thick line and thin line present the mean amplitude and SD (+/-) respectively. The time period 0-100 represent 0 to 180 degrees in the upstroke and 100-200 represents 180-0 degrees in the down stroke.

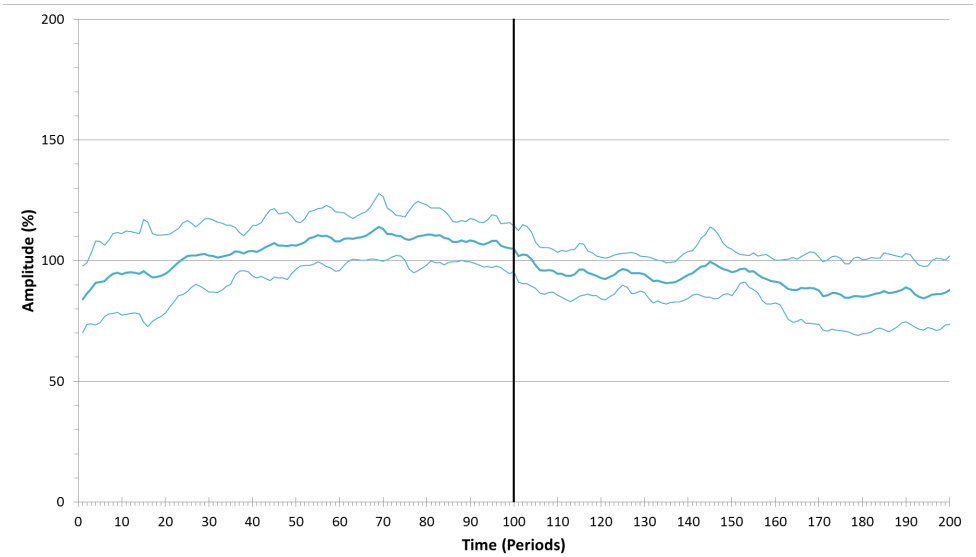

Figure A3.9. Graph to show the normal shoulder group (n=UT) activation for UT for the movement abduction/adduction whilst in the Supine position. The thick line and thin line present the mean amplitude and SD (+/-) respectively. The time period 0-100 represents the upstroke and 100-200 represents the down stroke.

Serratus Anterior

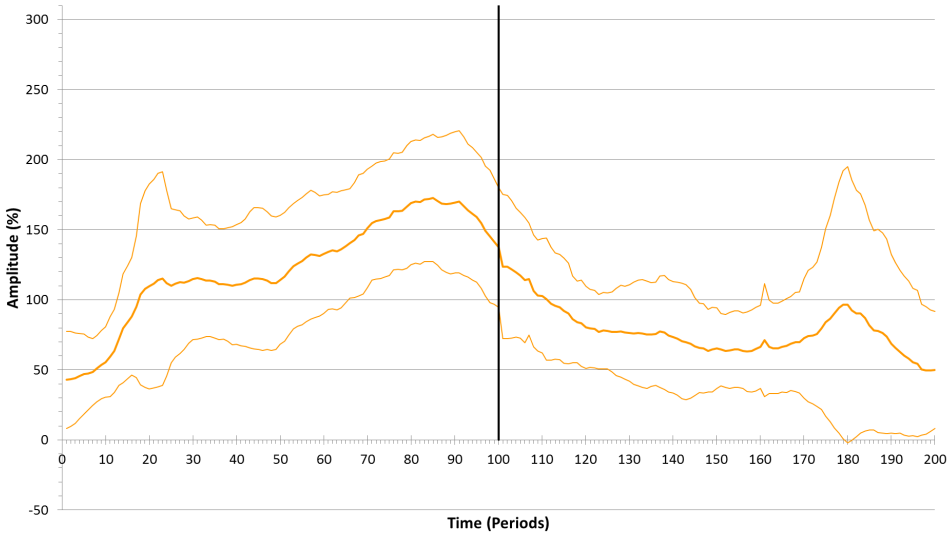

Figure A3.10. Graph to show the normal shoulder group (n=16) activation for SA for the movement abduction. The thick line and thin line present the mean amplitude and SD (+/-) respectively. The time period 0-100 represent 0 to 180 degrees in the upstroke and 100-200 represents 180-0 degrees in the down stroke.

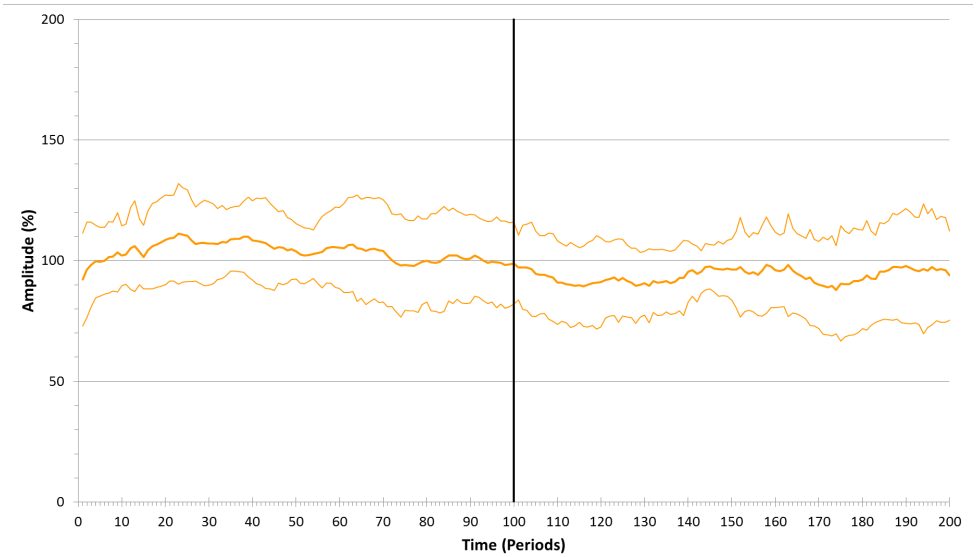

Figure A3.11. Graph to show the normal shoulder group (n=18) activation for SA for the movement abduction/addution whilst in the Supine position. The thick line and thin line present the mean amplitude and SD (+/-) respectively. The time period 0-100 represents the upstroke and 100-200 represents the down stroke.

Teres Major

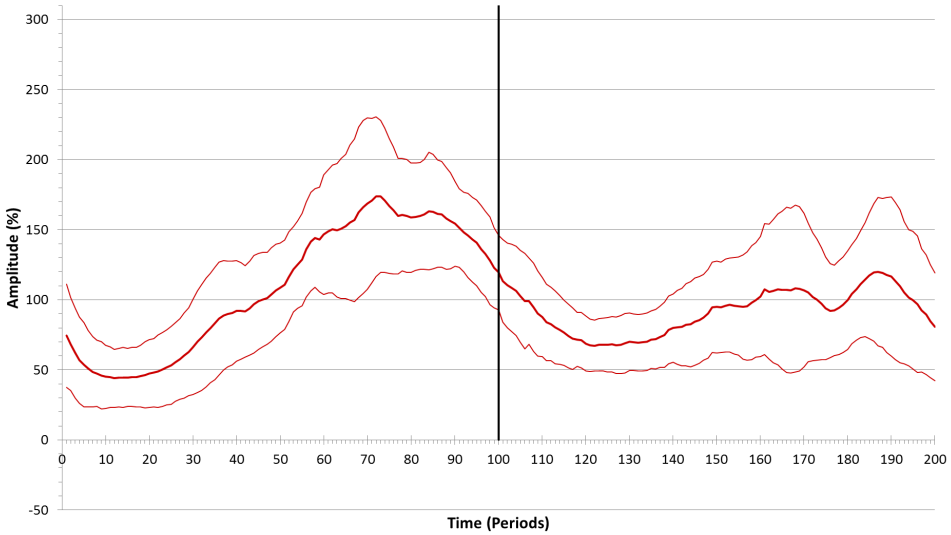

Figure A3.12. Graph to show the normal shoulder group (n=14) activation for TM for the movement abduction. The thick line and thin line present the mean amplitude and SD (+/-) respectively. The time period 0-100 represent 0 to 180 degrees in the upstroke and 100-200 represents 180-0 degrees in the down stroke.

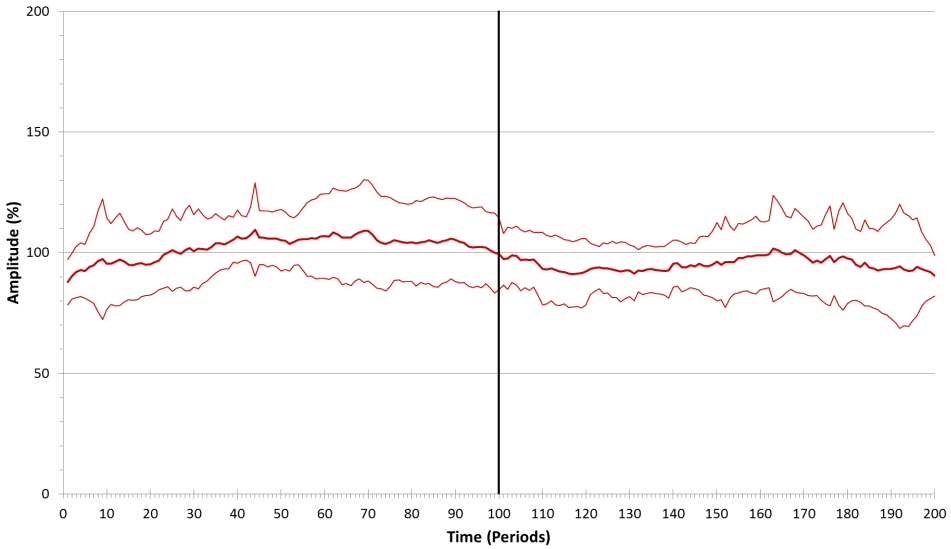

Figure A3.13. Graph to show the normal shoulder group (n=19) activation for TM for the movement abduction/adduction whilst in the Supine position. The thick line and thin line present the mean amplitude and SD (+/-) respectively. The time period 0-100 represents the upstroke and 100-200 represents the down stroke.

Latissimus Dorsi

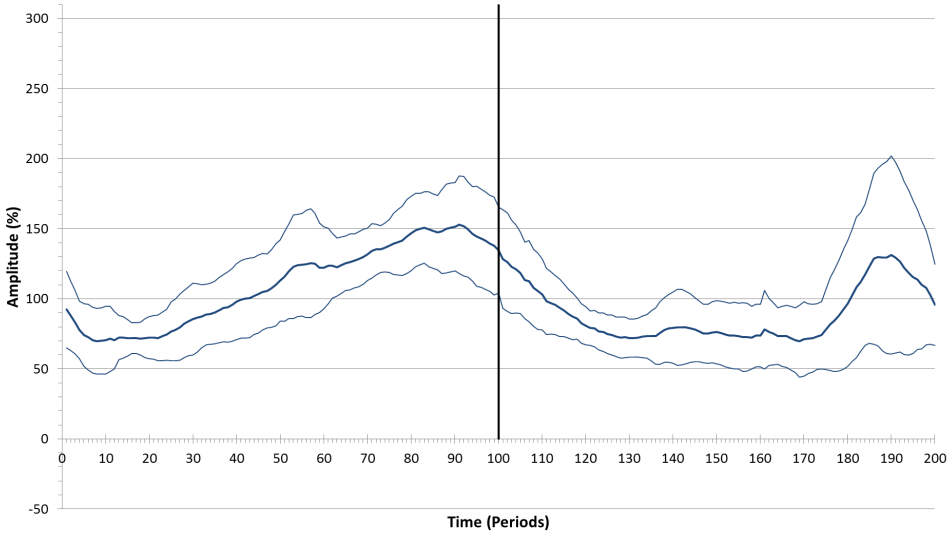

Figure A3.14. Graph to show the normal shoulder group (n=11) activation for LD for the movement abduction. The thick line and thin line present the mean amplitude and SD (+/-) respectively. The time period 0-100 represent 0 to 180 degrees in the upstroke and 100-200 represent 180-0 degrees in the down stroke.

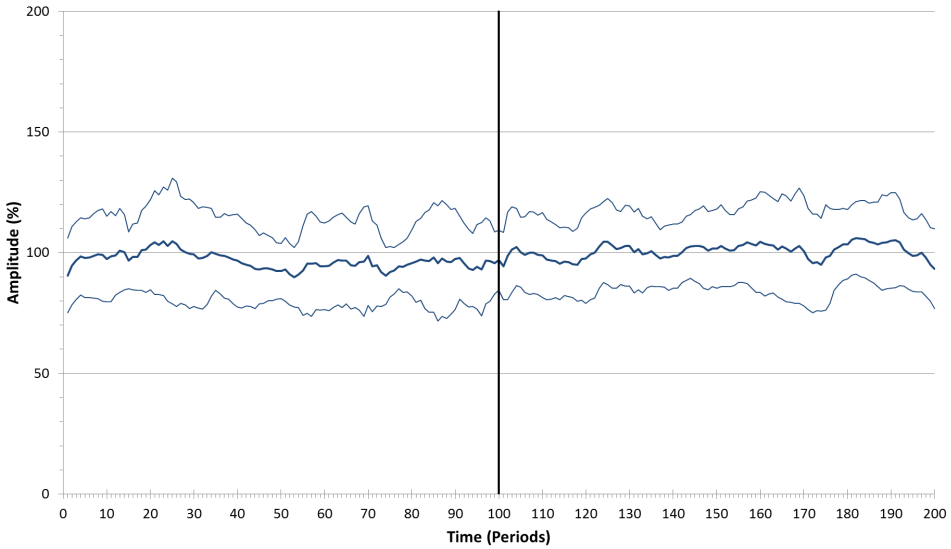

Figure A3.15. Graph to show the normal shoulder group (n=16) activation for LD for the movement abduction/addution whilst in the Supine position. The thick line and thin line present the mean amplitude and SD (+/-) respectively. The time period 0-100 represents the upstroke and 100-200 represents the down stroke.

Pectoralis Major

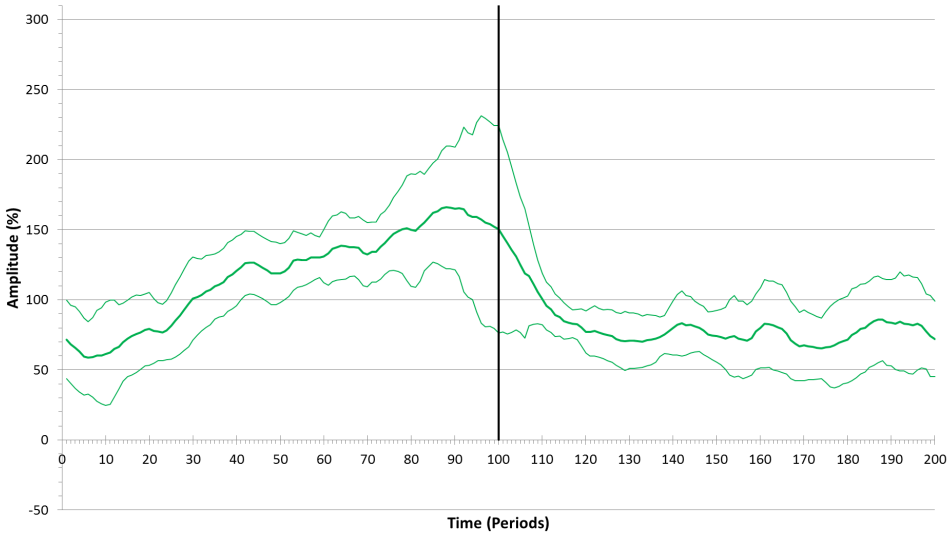

Figure A3.16. Graph to show the normal shoulder group (n=11) activation for PM for the movement abduction. The thick line and thin line present the mean amplitude and SD (+/-) respectively. The time period 0-100 represent 0 to 180 degrees in the upstroke and 100-200 represent 180-0 degrees in the down stroke.

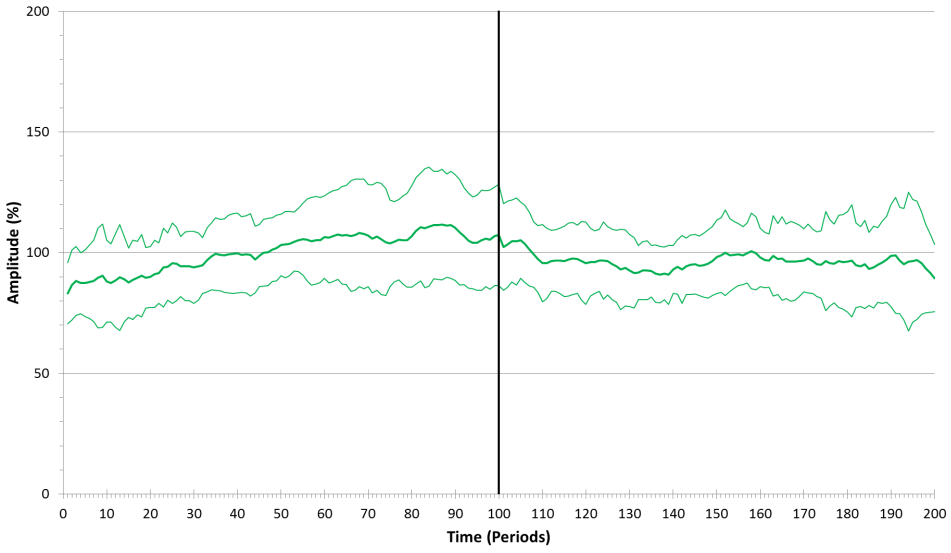

Figure A3.17. Graph to show the normal shoulder group (n=20) activation for PM for the movement abduction/addution whilst in the Supine position. The thick line and thin line present the mean amplitude and SD (+/-) respectively. The time period 0-100 represents the upstroke and 100-200 represents the down stroke.

Biceps Brachii

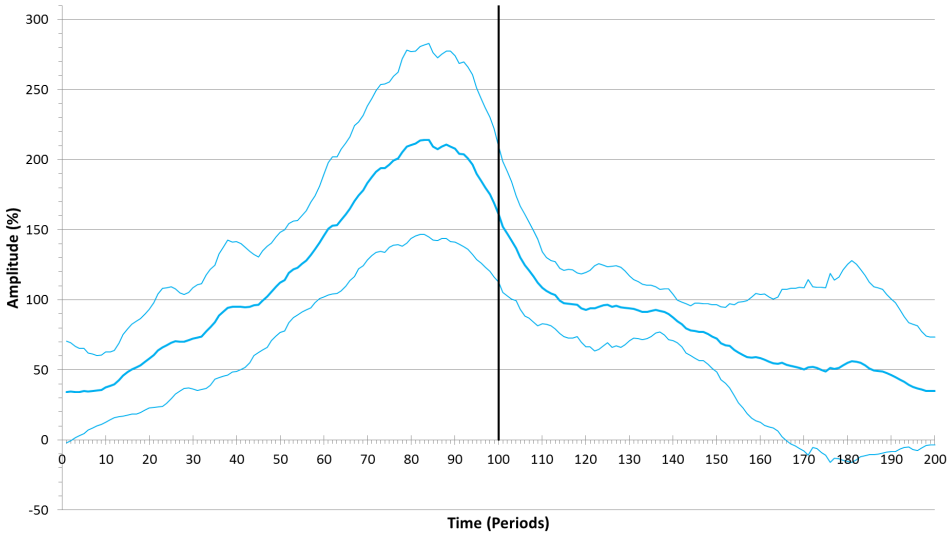

Figure A3.18. Graph to show the normal shoulder group (n=12) activation for BB for the movement abduction. The thick line and thin line present the mean amplitude and SD (+/-) respectively. The time period 0-100 represent 0 to 180 degrees in the upstroke and 100-200 represents 180-0 degrees in the down stroke.

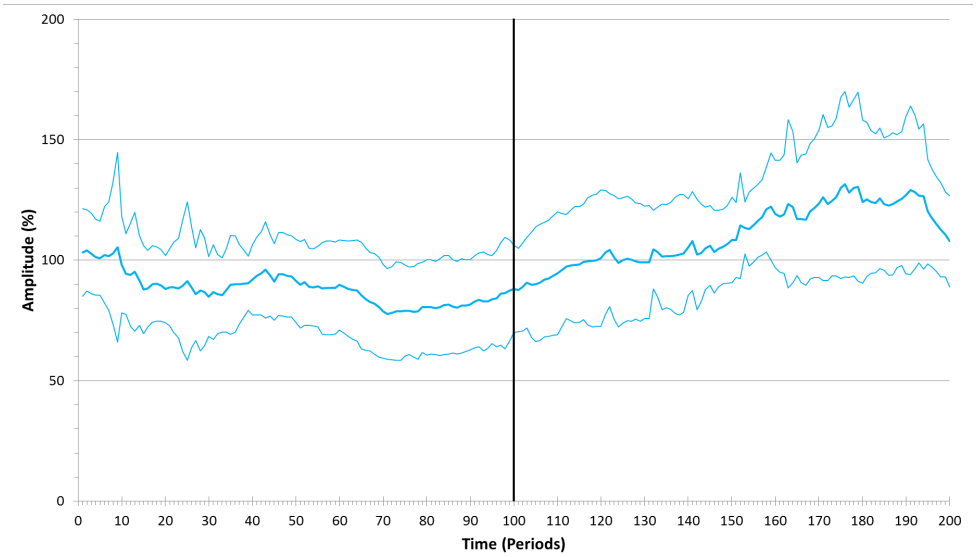

Figure A3.19. Graph to show the normal shoulder group (n=16) activation for BB for the movement abduction/addution whilst in the Supine position. The thick line and thin line present the mean amplitude and SD (+/-) respectively. The time period 0-100 represents the upstroke and 100-200 represents the down stroke.

Supraspinatus

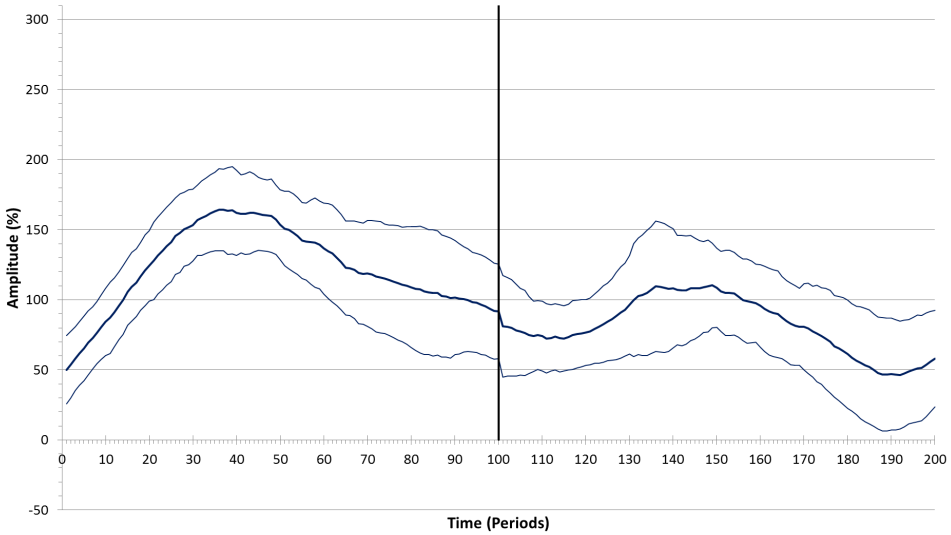

Figure A3.20. Graph to show the normal shoulder group (n=17) activation for SSP for the movement abduction. The thick line and thin line present the mean amplitude and SD (+/-) respectively. The time period 0-100 represent 0 to 180 degrees in the upstroke and 100-200 represent 180-0 degrees in the down stroke.

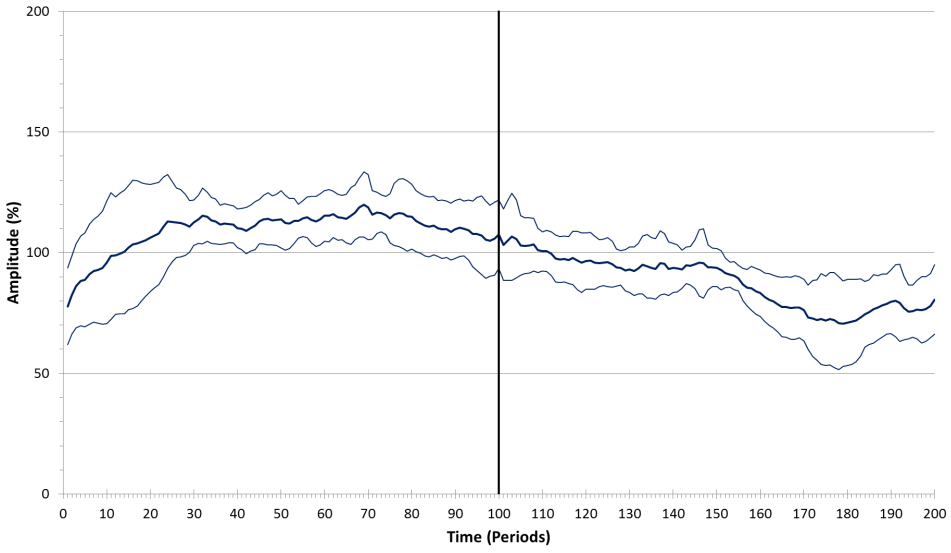

Figure A3.21. Graph to show the normal shoulder group (n=11) activation for SSP for the movement abduction/addution whilst in the Supine position. The thick line and thin line present the mean amplitude and SD (+/-) respectively. The time period 0-100 represents the upstroke and 100-200 represents the down stroke.

Infraspinatus

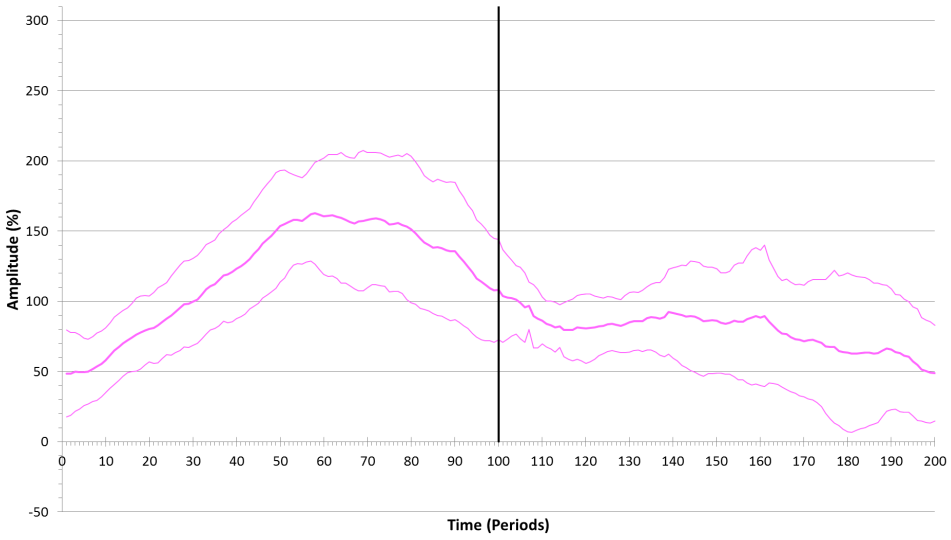

Figure A3.22. Graph to show the normal shoulder group (n=13) activation for ISP for the movement abduction. The thick line and thin line present the mean amplitude and SD (+/-) respectively. The time period 0-100 represent 0 to 180 degrees in the upstroke and 100-200 represents 180-0 degrees in the down stroke.

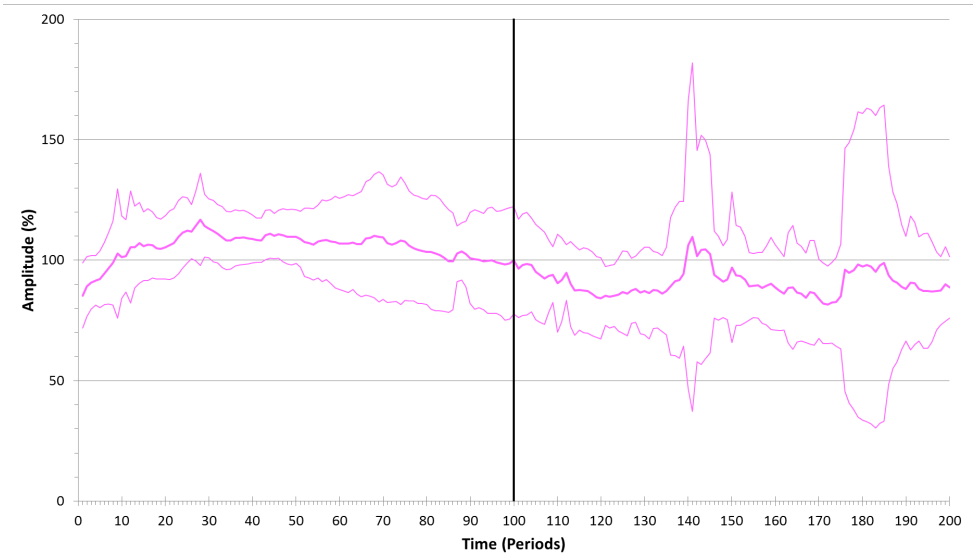

Figure A3.23. Graph to show the normal shoulder group (n=17) activation for ISP for the movement abduction/addution whilst in the Supine position. The thick line and thin line present the mean amplitude and SD (+/-) respectively. The time period 0-100 represents the upstroke and 100-200 represents the down stroke.

Subscapularis

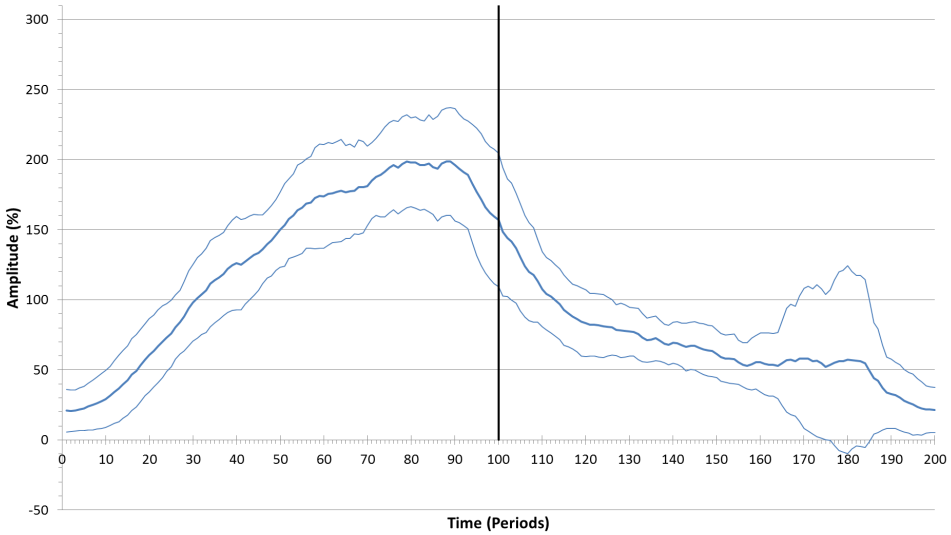

Figure A3.24. Graph to show the normal shoulder group (n=8) activation for SUB for the movement abduction. The thick line and thin line present the mean amplitude and SD (+/-) respectively. The time period 0-100 represent 0 to 180 degrees in the upstroke and 100-200 represent 180-0 degrees in the down stroke.

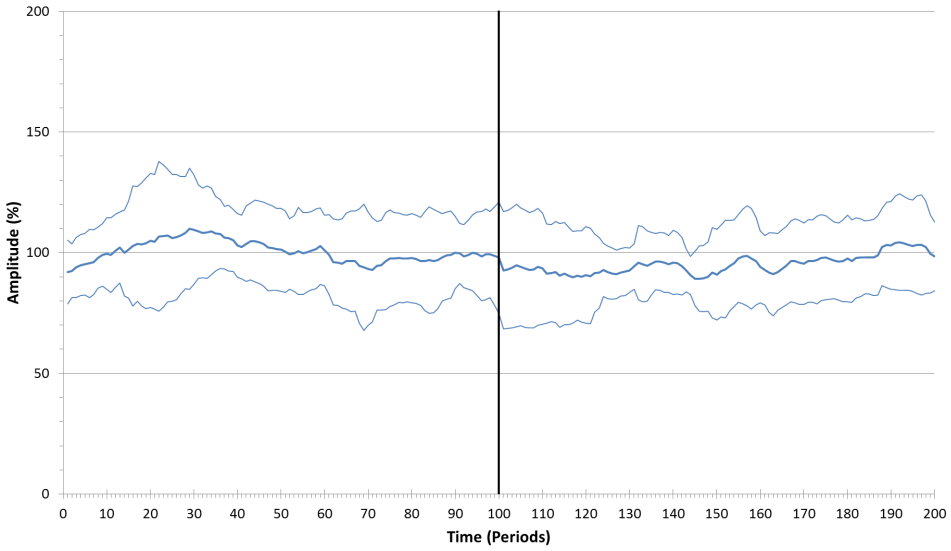

Figure A3.25. Graph to show the normal shoulder group (n=11) activation for AD for the movement abduction/addution whilst in the Supine position. The thick line and thin line present the mean amplitude and SD (+/-) respectively. The time period 0-100 represents the upstroke and 100-200 represents the down stroke.

Appendix 4 – EMG - Comparative Study - Abduction

Anterior Deltoid

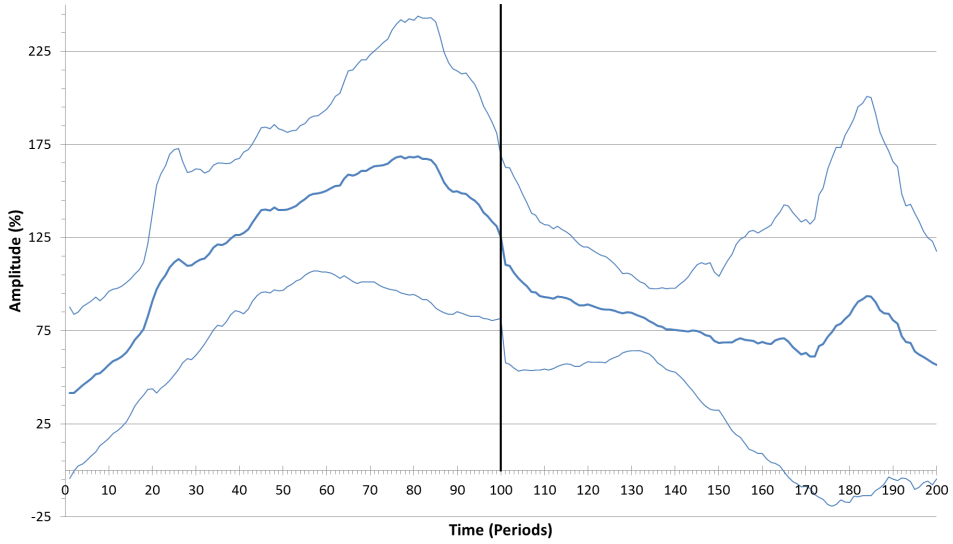

Figure A4.1. Graph to show the Patient group (n=12) activation for AD for the movement adduction/abduction. The thick line and thin line present the mean amplitude and SD (+/-) respectively. The time period 0-100 represent 0 to 90 degrees in the upstroke and 100-200 represent 90-0 degrees in the down stroke.

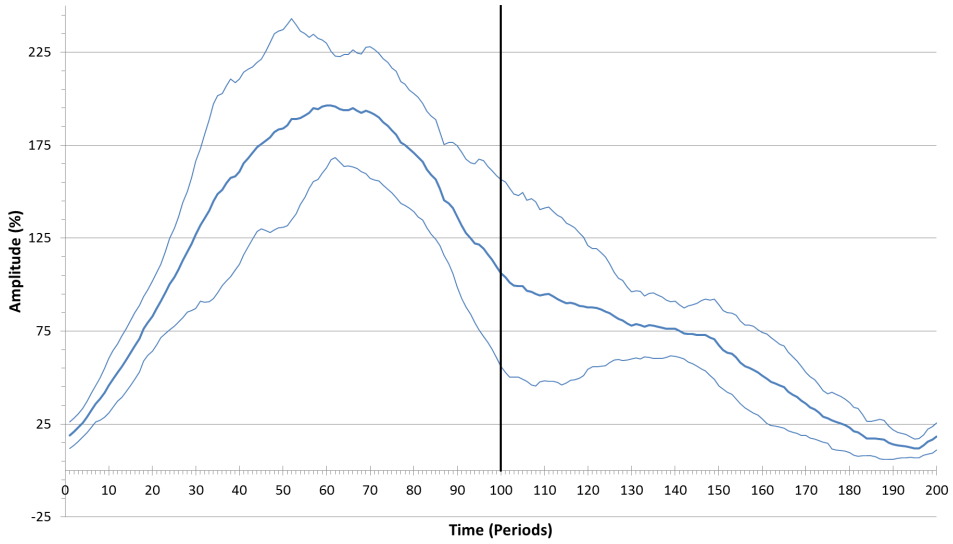

Figure A4.2. Graph to show the Control group (n=12) activation for AD for the movement adduction/abduction. The thick line and thin line present the mean amplitude and SD (+/-) respectively. The time period 0-100 represent 0 to 90 degrees in the upstroke and 100-200 represents 90-0 degrees in the down stroke.

Middle Deltoid

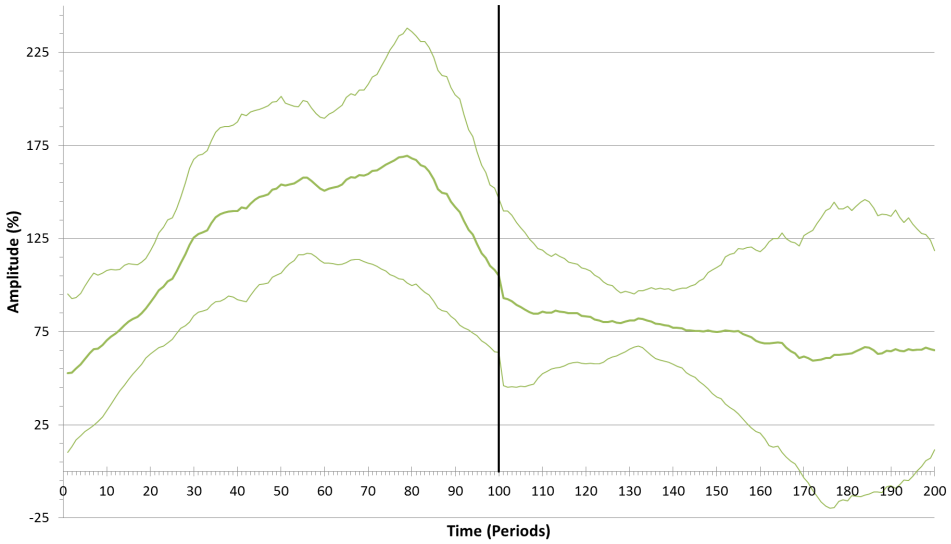

Figure A4.3. Graph to show the Patient group (n=12) activation for MD for the movement adduction/abduction. The thick line and thin line present the mean amplitude and SD (+/-) respectively. The time period 0-100 represent 0 to 90 degrees in the upstroke and 100-200 represents 90-0 degrees in the down stroke.

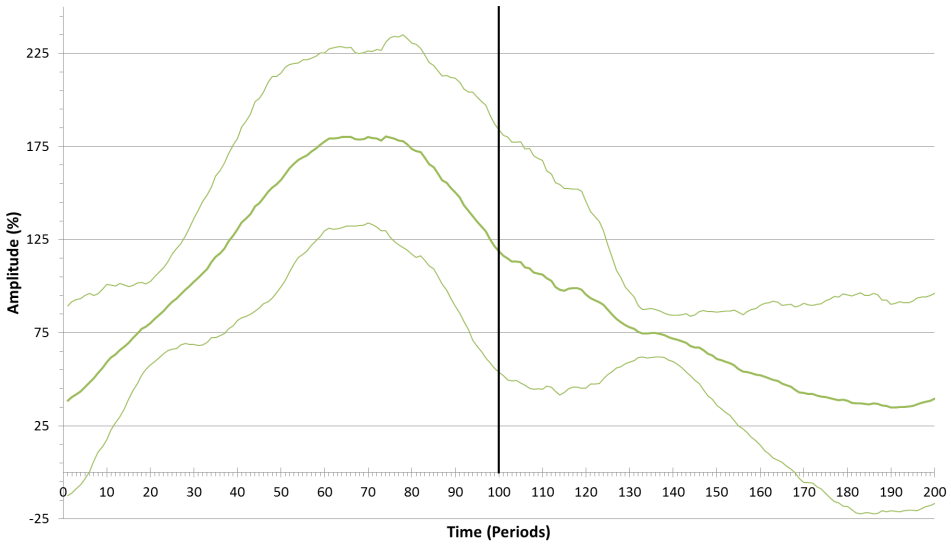

Figure A4.4. Graph to show the Control group (n=13) activation for MD for the movement adduction/abduction. The thick line and thin line present the mean amplitude and SD (+/-) respectively. The time period 0-100 represent 0 to 90 degrees in the upstroke and 100-200 represents 90-0 degrees in the down stroke.

Posterior Deltoid

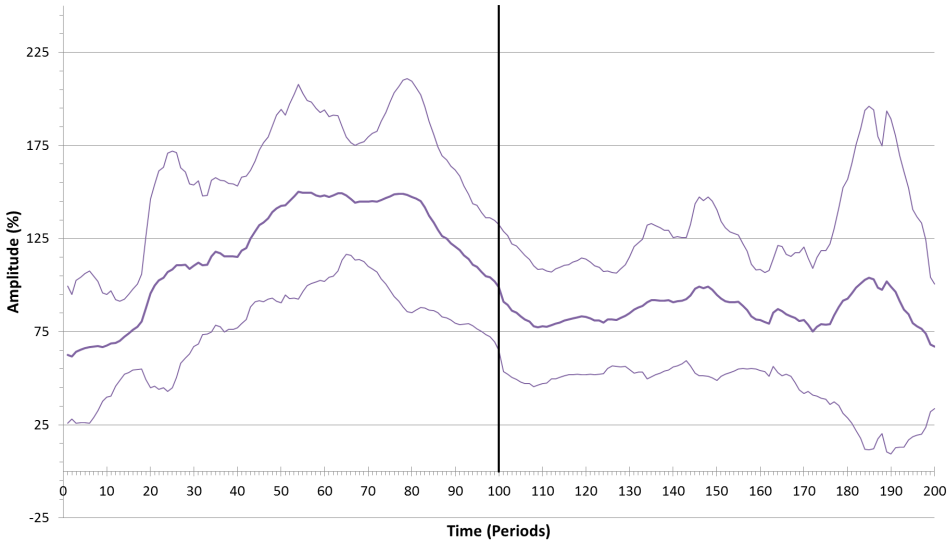

Figure A4.5. Graph to show the Patient group (n=12) activation for PD for the movement adduction/abduction. The thick line and thin line present the mean amplitude and SD (+/-) respectively. The time period 0-100 represent 0 to 90 degrees in the upstroke and 100-200 represents 90-0 degrees in the down stroke.

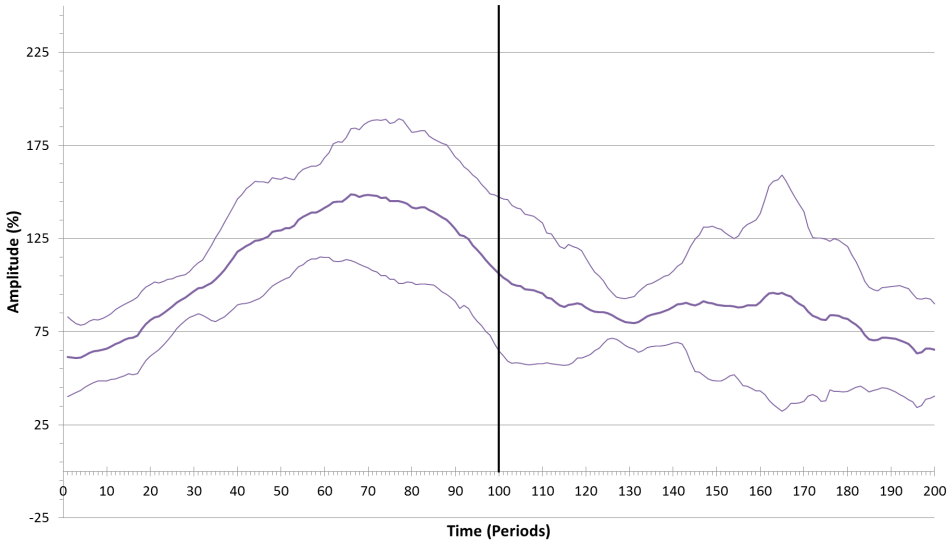

Figure A4.6. Graph to show the Patient group (n=12) activation for PD for the movement adduction/abduction. The thick line and thin line present the mean amplitude and SD (+/-) respectively. The time period 0-100 represent 0 to 90 degrees in the upstroke and 100-200 represents 90-0 degrees in the down stroke.

Upper Trapezium

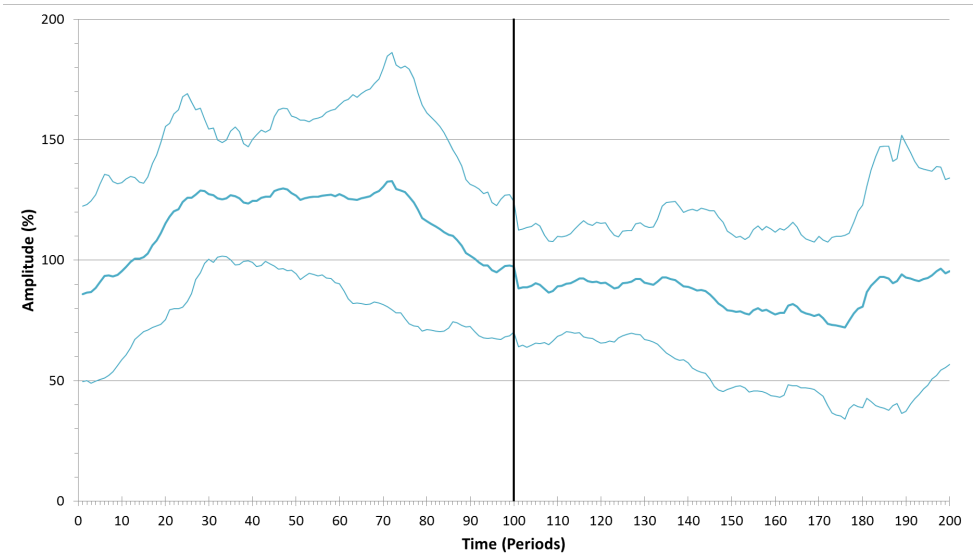

Figure A4.7. Graph to show the Patient group (n=12) activation for UT for the movement adduction/abduction. The thick line and thin line present the mean amplitude and SD (+/-) respectively. The time period 0-100 represent 0 to 90 degrees in the upstroke and 100-200 represents 90-0 degrees in the down stroke.

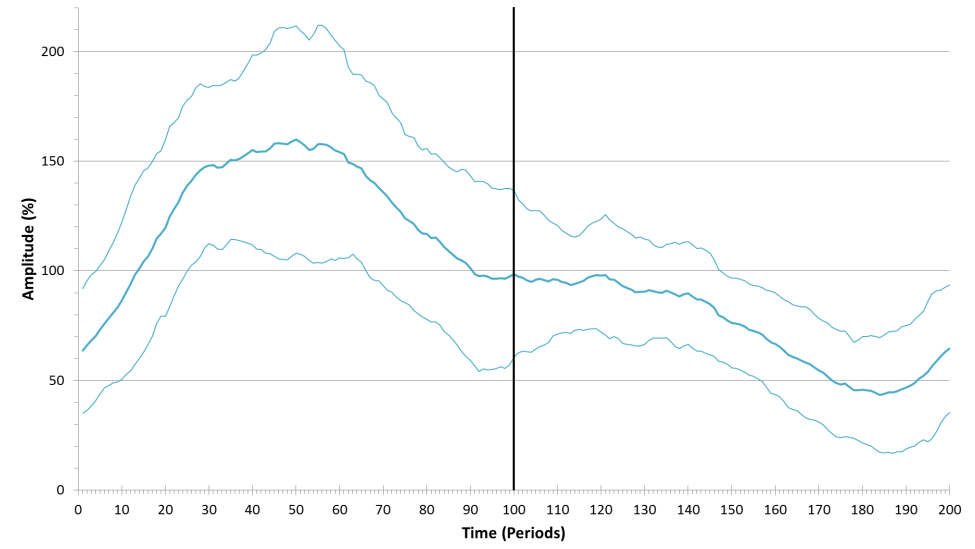

Figure A4.8. Graph to show the Control group (n=13) activation for UT for the movement adduction/abduction. The thick line and thin line present the mean amplitude and SD (+/-) respectively. The time period 0-100 represent 0 to 90 degrees in the upstroke and 100-200 represents 90-0 degrees in the down stroke.

Serratus Anterior

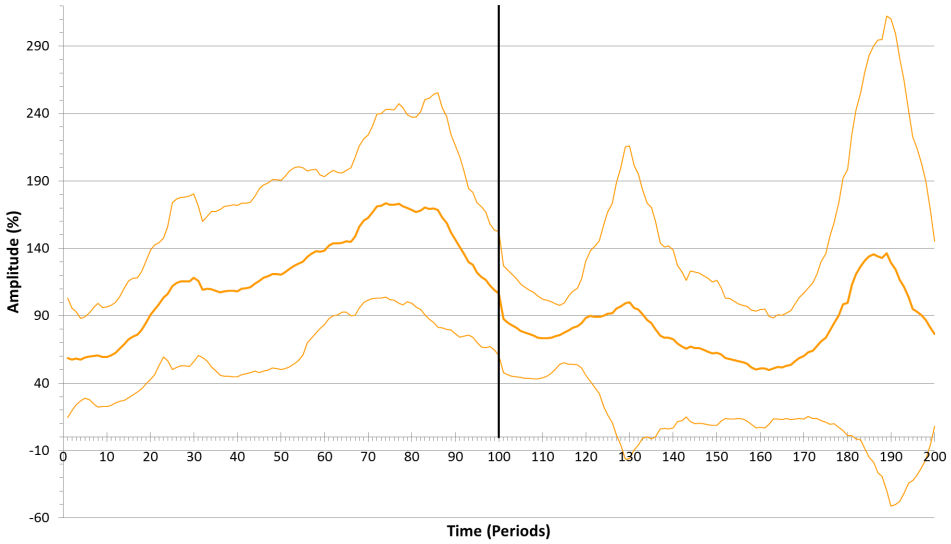

Figure A4.9. Graph to show the Patient group (n=11) activation for SA for the movement adduction/abduction. The thick line and thin line present the mean amplitude and SD (+/-) respectively. The time period 0-100 represent 0 to 90 degrees in the upstroke and 100-200 represents 90-0 degrees in the down stroke.

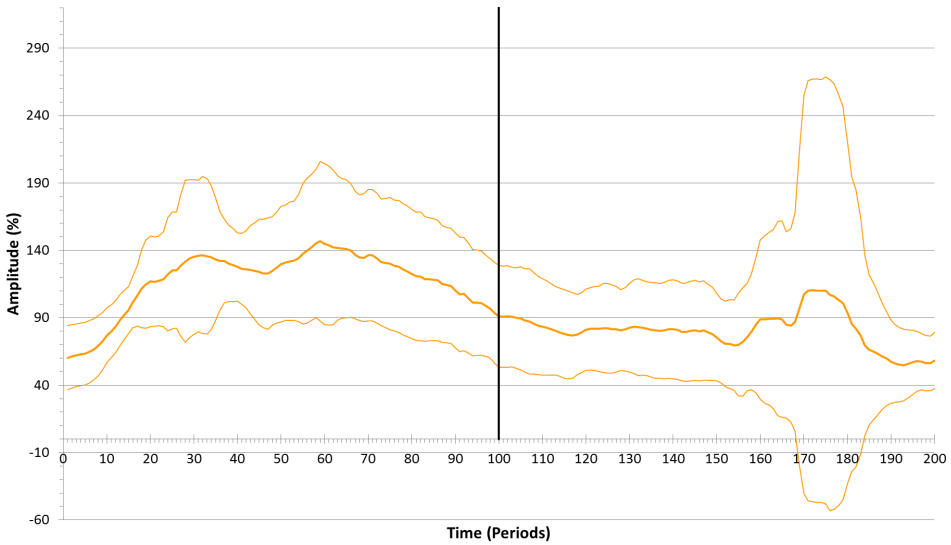

Figure A4.10. Graph to show the Control group (n=10) activation for SA for the movement adduction/abduction. The thick line and thin line present the mean amplitude and SD (+/-) respectively. The time period 0-100 represent 0 to 90 degrees in the upstroke and 100-200 represents 90-0 degrees in the down stroke.

Teres Major

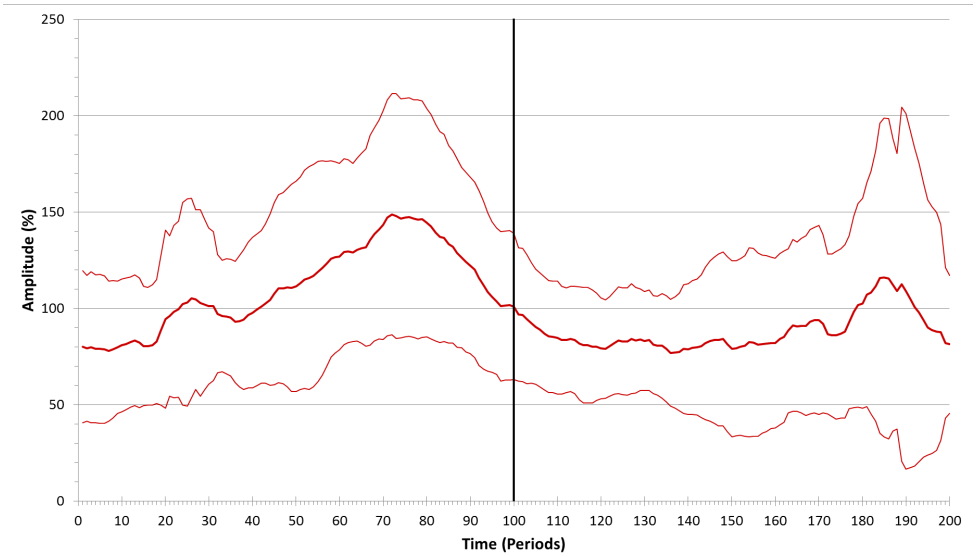

Figure A4.11. Graph to show the Patient group (n=11) activation for TM for the movement adduction/abduction. The thick line and thin line present the mean amplitude and SD (+/-) respectively. The time period 0-100 represent 0 to 90 degrees in the upstroke and 100-200 represents 90-0 degrees in the down stroke.

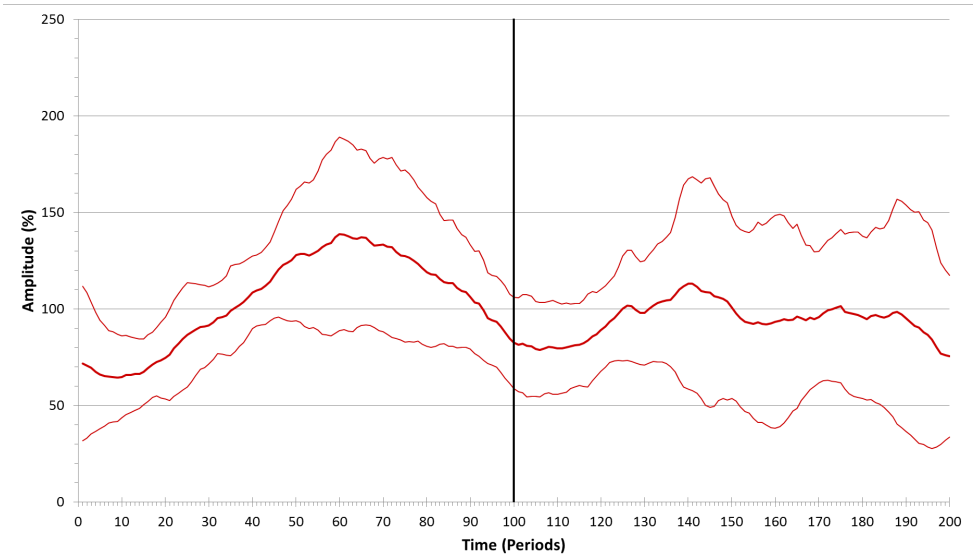

Figure A4.12. Graph to show the Control group (n=8) activation for TM for the movement adduction/abduction. The thick line and thin line present the mean amplitude and SD (+/-) respectively. The time period 0-100 represent 0 to 90 degrees in the upstroke and 100-200 represents 90-0 degrees in the down stroke.

Latissimus Dorsi

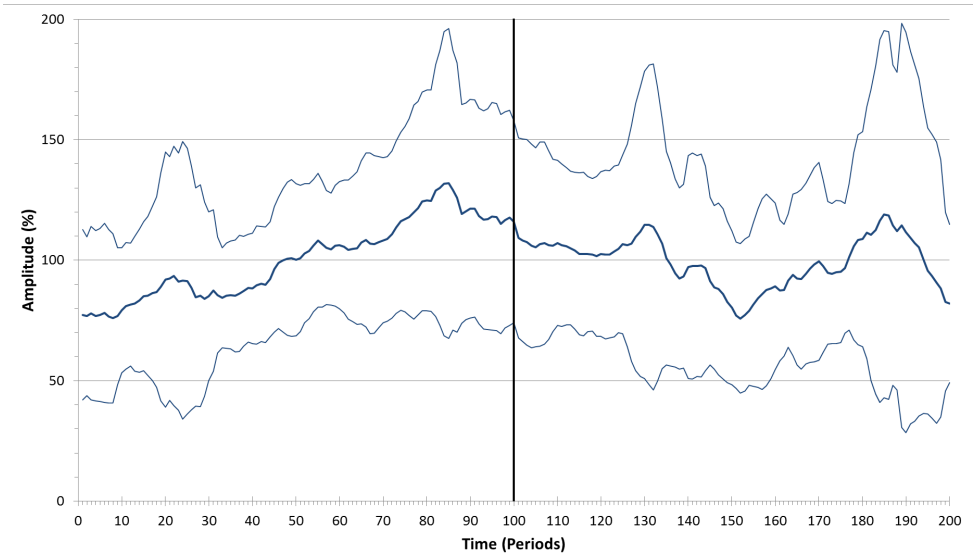

Figure A4.13. Graph to show the Patient group (n=12) activation for LD for the movement adduction/abduction. The thick line and thin line present the mean amplitude and SD (+/-) respectively. The time period 0-100 represent 0 to 90 degrees in the upstroke and 100-200 represents 90-0 degrees in the down stroke.

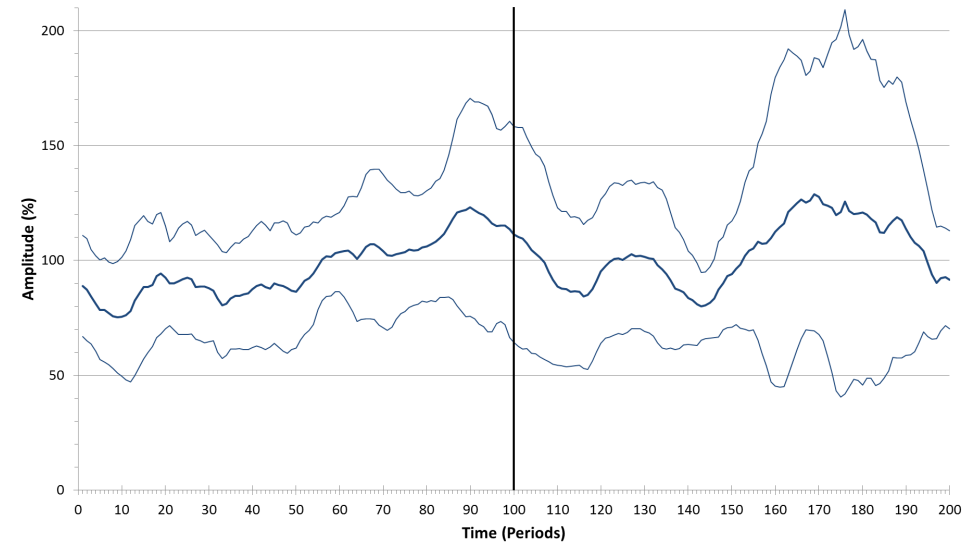

Figure A4.14. Graph to show the Control group (n=9) activation for LD for the movement adduction/abduction. The thick line and thin line present the mean amplitude and SD (+/-) respectively. The time period 0-100 represent 0 to 90 degrees in the upstroke and 100-200 represents 90-0 degrees in the down stroke.

Pectoralis Major

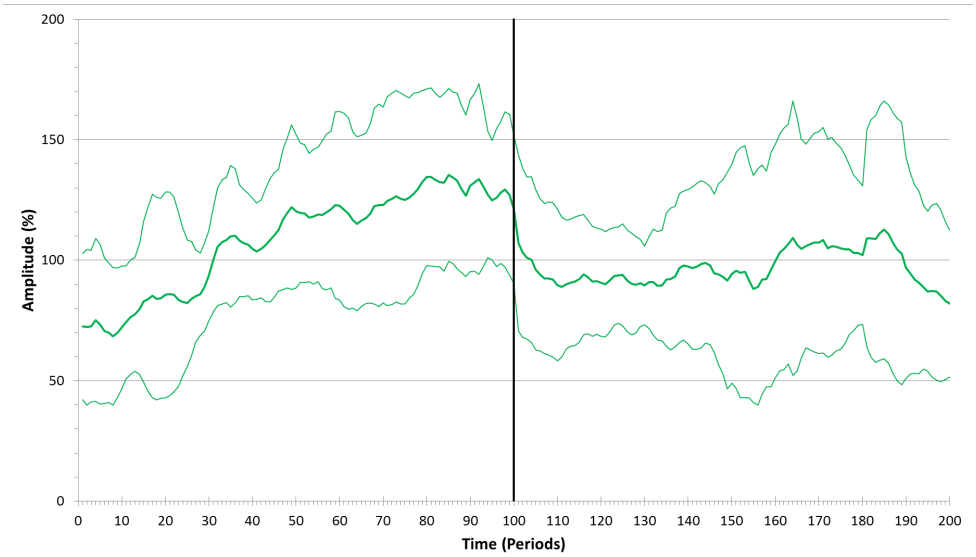

Figure A4.15. Graph to show the Patient group (n=12) activation for PM for the movement adduction/abduction. The thick line and thin line present the mean amplitude and SD (+/-) respectively. The time period 0-100 represent 0 to 90 degrees in the upstroke and 100-200 represents 90-0 degrees in the down stroke.

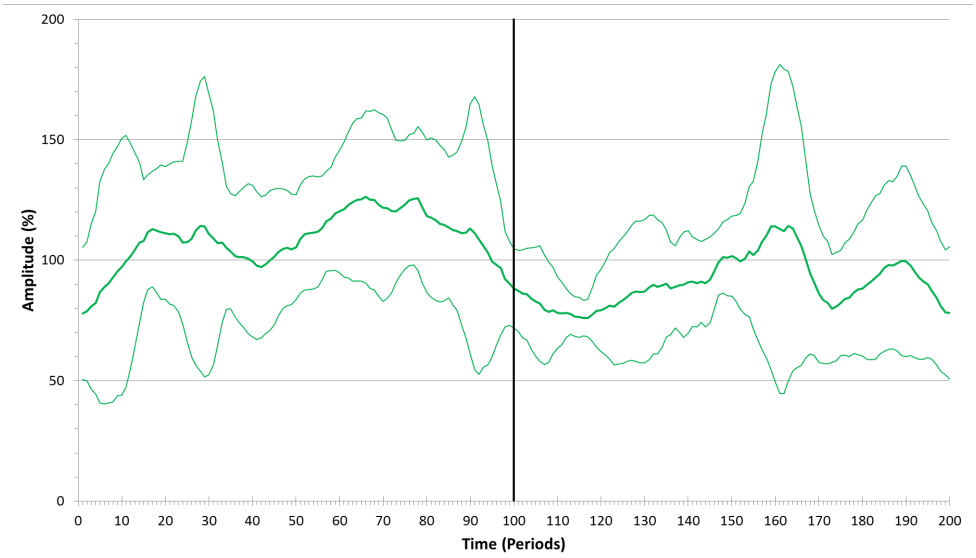

Figure A4.16. Graph to show the Control group (n=9) activation for PM for the movement adduction/abduction. The thick line and thin line present the mean amplitude and SD (+/-) respectively. The time period 0-100 represent 0 to 90 degrees in the upstroke and 100-200 represents 90-0 degrees in the down stroke.

Biceps Brachii

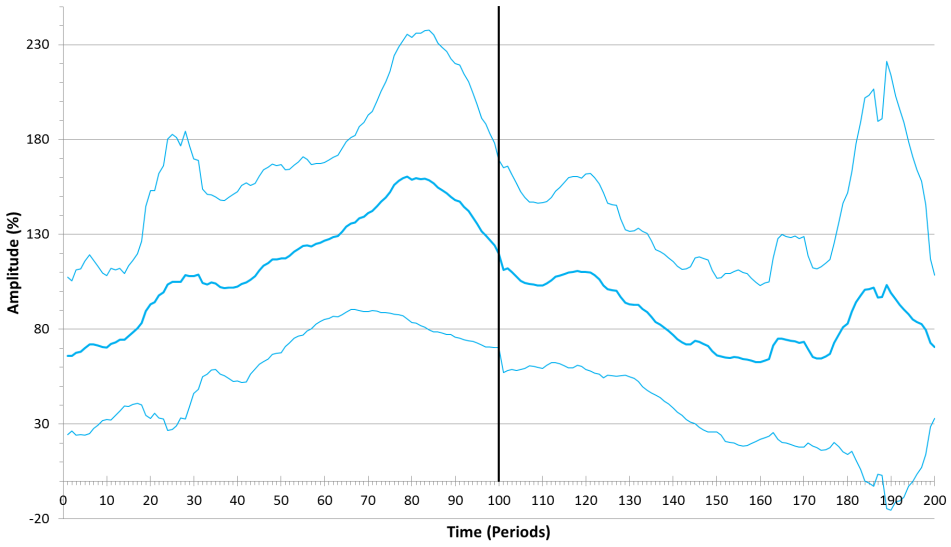

Figure A4.18. Graph to show the Patient group (n=12) activation for BB for the movement adduction/abduction. The thick line and thin line present the mean amplitude and SD (+/-) respectively. The time period 0-100 represent 0 to 90 degrees in the upstroke and 100-200 represents 90-0 degrees in the down stroke.

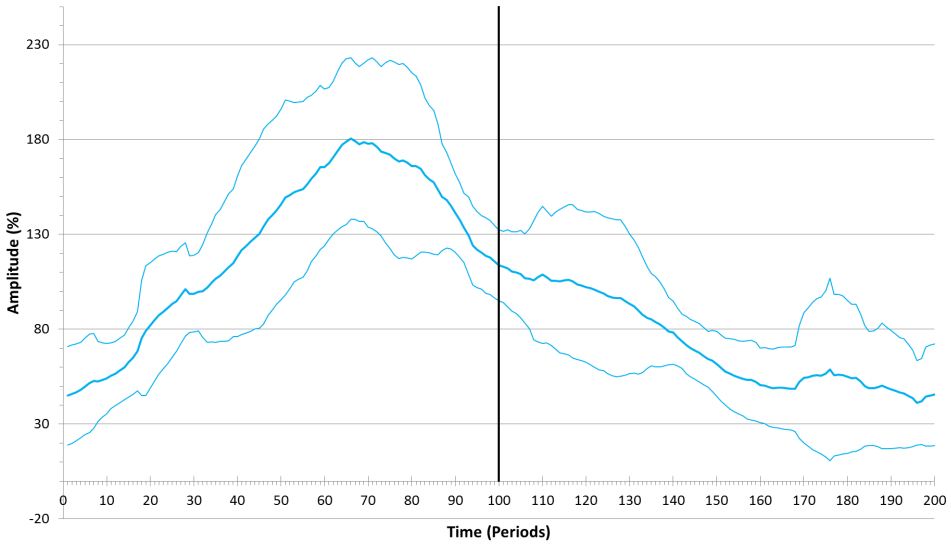

Figure A4.19. Graph to show the Control group (n=10) activation for BB for the movement adduction/abduction. The thick line and thin line present the mean amplitude and SD (+/-) respectively. The time period 0-100 represent 0 to 90 degrees in the upstroke and 100-200 represents 90-0 degrees in the down stroke.

Infraspinatus

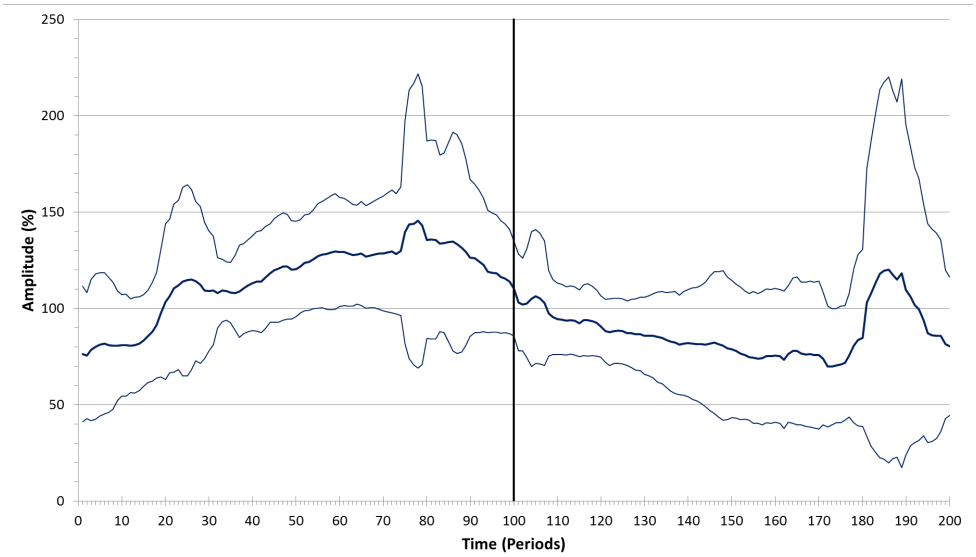

Figure A4.20. Graph to show the Patient group (n=12) activation for ISP for the movement adduction/abduction. The thick line and thin line present the mean amplitude and SD (+/-) respectively. The time period 0-100 represent 0 to 90 degrees in the upstroke and 100-200 represents 90-0 degrees in the down stroke.

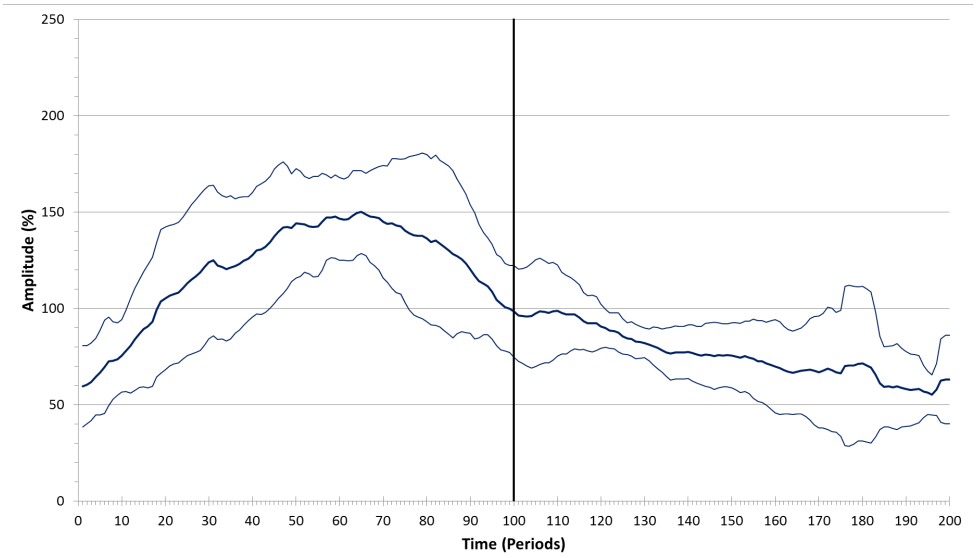

Figure A4.21. Graph to show the Control group (n=11) activation for ISP for the movement adduction/abduction. The thick line and thin line present the mean amplitude and SD (+/-) respectively. The time period 0-100 represent 0 to 90 degrees in the upstroke and 100-200 represents 90-0 degrees in the down stroke.

Appendix 5 – Participant Information Sheet

Simon P Frostick, MA DM FRCS

Professor of Orthopaedics

Musculoskeletal Science Research Group

Division of Surgery and Oncology

School of Cancer Studies

Royal Liverpool University Hospital

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Email: s.p.frostick@liv.ac.uk

Patient Information Sheet: Control Group

Study Title: Functional pathophysiology of the shoulder girdle

You are being invited to take part in this research study. The following information is to help you to decide whether to take part in this study or not. Please take time to read this information carefully. You are free to discuss this information with your family doctor, your family and friends before you make a decision. Please ask us, one of the doctors responsible for running the study, if there is anything that you do not understand or if you would like to obtain some more information. Thank you for reading this.

Purpose of the study

Background and Aims:

We use our arms for an amazing range of activities; from washing and feeding ourselves to playing tennis and using a computer. In order for us to perform these tasks easily, all parts of the arm must work normally. In other words, the bones, ligaments, nerves, muscles and tendons of the shoulder and upper arm must work properly. Any loss of movement in the shoulder will limit what we can do. For example, if we cannot lift our arm forward to the level of our shoulder then we may not be able to reach a shelf. Loss of function around the shoulder from whatever cause results in varying levels of physical impairment and disability. In older people this can mean that they may no longer look after themselves. In younger people they may not be able to work properly.

Shoulder/arm movements are complicated. In the normal shoulder various muscles work together in a coordinated pattern in order to move the head of the humerus bone against the shoulder joint socket (the glenoid). Sensory structures

within the joint capsule (that surrounds the joint) and in the muscles and tendons of the joint constantly send feedback information to tell you where your arm is, which muscles are working and give warnings to prevent injury. If you have a problem in your shoulder this complex coordination may be affected, damaging the shoulder. In some people changes in some of the bones or tendons may cause the muscles to work poorly different from their usual coordinated fashion.

We are interested in studying 3 shoulder problems. The first is called shoulder instability where the shoulder has a tendency to come out of joint (subluxate or dislocate); this is usually found in younger people. The second called impingement (sub-acromial impingement syndrome) is found mostly in middle aged people and is a major cause of shoulder pain and restricted movement; we do not know the cause of this problem. The last is where some of the tendons that help to lift the arm up become torn (a rotator cuff tear). This occurs mostly in older people and half the people over 80 years may be affected. Each of these problems causes major difficulties in shoulder function for the sufferers. Instability can be so bad that the shoulder dislocates whenever the arm is moved. In impingement syndrome the pain and stiffness can prevent the person doing very simple tasks such as washing their hair or dressing. Older people with a rotator cuff tear may not be able to lift their arm at all.

In all 3 conditions the same muscles, tendons, ligaments, capsule and bones are involved and it seems likely that there are common underlying problems and features. We would like to use the same investigations to find out what is going on in all 3 shoulder problems and compare the results between the three shoulder problems and with those from people with normal shoulder function.

This will allow us to standardise the assessments, identify common features and develop new ideas about treatment.

We are also interested in studying the contribution of shoulder girdle muscles into recommended upper extremity rehabilitation exercises. This will help to rationalise the exercise prescription with reference to a given shoulder pathology.

Who is organising the study?

This study is organised by the Musculoskeletal Science Research Group at the Royal Liverpool University Hospital. The doctor responsible for the overall conduct of this study is professor Simon Frostick (0151 706 4120). Our group has a lot of experience in carrying out research on shoulder problems as well as treating people with these conditions. This study will be carried out in accordance with Good Clinical Practice Guidelines.

Why have I been chosen?

You have no problem with either of your shoulders now nor do you have a history of shoulder problems. We will be asking upper limb patients to undertake the variety of tests described below. We would like to ask you to undertake exactly the same series of tests to provide the standards against which the function of patients with problems can be measured. In this way we will be able to identify and understand their problems better.

What does this study involve?

We would like to ask for your permission to carry out several different aspects of the study which will include completing some questionnaires, measuring the electrical activity (electromyography) and strength of some muscles around your shoulder, looking at how you control your muscles, measuring the water and fat content of your muscles, measuring the pressure within your muscles, and taking some blood for laboratory tests. Electromyography and pressure measurement of some muscles will involve using needles and fine wires.

We will ask you to consent to each part of this research project separately, so that if there is any part you are unhappy about you need not consent to it.

If you do decide to take part, you will be given this information sheet to keep and a consent form. Even if you decide to take part, you are still free to withdraw at any time and without giving a reason. It is up to you whether you wish to take part in this study.

What will happen to me if I take part?

1. Questionnaires:

- This will involve completing a number of simple questionnaires and being examined by a member of the team.
- If you need assistance in completing the questionnaires we will provide help. If you have treatment, we would like to repeat these aspects of the study before your treatment and then at 6 weeks, 3 months, 6 months, 12 months and 24

months after your operation. You will not be expected to come to the clinic any more times than you would if you were not part of this research project. These assessments are routinely used in many clinics. It will take about 30-45 minutes to complete the questionnaires.

- We will keep all the results of the questionnaires in a booklet that is separate to your normal hospital case sheet. We will also store the results of the questionnaires on a computer linked to the NHS computer network. We will put a summary of the results into your hospital notes so that anyone needing to access your medical records can see how you are doing as far as your shoulder is concerned. This is useful information for the doctors and physiotherapists who will see you for your routine follow-up in the clinic.

2. Electromyography and strength measurements:

- We would like to study the strength and activity patterns of your shoulder muscles during some upper extremity movements, tasks, and exercises. We will place electrodes on your skin over some of the muscles and use special instruments to measure the electrical activity generated by the muscles (electromyography or EMG). In two more deeply located muscles we will place a fine-wire electrode into the muscle using a small needle: we have a lot of years of experience of doing this, the needles are disposable and will be put in with a proper sterile technique. This procedure may cause minor discomfort but we can use a local anaesthetic if you prefer. We will also measure how much strength you have in some of your shoulder and arm muscles by getting you to either pull on or squeeze a machine called a dynamometer.

3. Motion Analysis

- We will use a technique called motion analysis or motion capture to see how you move upper extremity and how you control pelvis and trunk (core) during different tasks which involve the use of your arm and shoulder. With markers placed on standard landmarks of the joints, this non-invasive technique uses 3-dimensional motion capture cameras to track the motion of your joints, trunk and pelvis. Sometimes we are also interested to perform this measurement inside an MRI scanner whilst you are doing simple tasks with your arm, in this case we will provide you with additional information.

4. Blood sample

- In addition to routine blood samples, we would like to take one sample collected into two different tubes for research. The blood sample will allow us to look at some chemicals in the blood that relate to your damaged tissues. Therefore, there will be 1 extra needle stick to obtain the blood sample. You might feel some mild discomfort from the needle or have some minor bruising around the site which should disappear in a few days. A total of 10ml (2x5ml) of your blood will be taken and stored for subsequent analysis. We give the sample a unique number which is linked to your name but the persons undertaking the laboratory analysis will not be able to identify you.

5. Bio-impedance analysis

- This technique will allow us to measure the percentage of fat in your body and the amount of muscle in your arm. A series of measuring sensors are put onto the skin and you will not feel anything during this very quick procedure.

6. Intramuscular pressure

- We would like to measure the pressure within some of the muscles of the shoulder. Some local anaesthetic is applied to the skin and a small sterile needle is put into the muscle to measure the pressure. This will tell us whether the muscles are able to keep their blood flow properly during activity. These measurements take a few minutes to complete.

Are there any advantages or disadvantages of taking part in this study?

If you decide to take part in this study it may not benefit you directly. The information obtained from this research, however, will give more information to scientists and may help doctors manage patients with soft tissue shoulder problems more effectively in the future.

Confidentiality – who will know I am taking part in the study?

All information which is collected about you during the course of the research will be kept strictly confidential. Any information about you which leaves the hospital will be anonymised so that you cannot be recognised from it. We may look at medical notes of some patients in order to track the history of shoulder problem and co-existing medical conditions that may affect shoulder performance. This will be done only by the research team members who have been the licence by the Royal Liverpool University and Broadgreen Hospitals NHS Trust to have controlled access to patient notes.

What will happen to the results of the study?

All the results of the study will be collected and submitted for publication in a medical journal. No personal details from which you could be identified will be included in any publications on the findings of this research.

What happens in the future?

Your treatment will continue to follow best practice guidelines for your particular problem. If new information comes to light during your treatment you will be informed and advised accordingly. We would be most grateful if you would agree to allow us to undertake additional related studies using your data if that should be appropriate. All such studies would be strictly anonymised and coded.

What if something goes wrong?

If you are harmed by taking part in this research project, there are no special compensation arrangements. If you are harmed due to someone's negligence, then you may have grounds for a legal action but you may have to pay for it. Regardless of this, if you wish to complain about any aspect of the way you have been approached or treated during the course of this study the normal National Health Service complaints mechanisms may be available to you.

The Liverpool Research Ethics Committee have reviewed this project and given their approval. If you have any queries you can contact

Appendix 6 – Consent Form

Simon P Frostick, MA DM FRCS
Professor of Orthopaedics

Musculoskeletal Science Research Group
Division of Surgery and Oncology
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Royal Liverpool University Hospital
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Trust Study No: 3744

CONSENT FORM: Functional Magnetic Resonance Imaging (fMRI)
Study Title: **Functional pathophysiology of the shoulder girdle**

Please initial box

1. I confirm that I have read and understand the patient information document For Trust Study Number 3744 (November 2008), for the study named above and that I have had the opportunity to ask questions. ☐
2. I understand that my participation is voluntary and that I am free to withdraw at any time without my medical care or legal rights being affected. ☐
3. I understand that sections of my medical notes may be looked at by individuals from the Royal Liverpool University and Broadgreen Hospitals NHS Trust. I give permission for these individuals to have access to my records. ☐

Functional Magnetic Resonance Imaging (fMRI) – I am happy to give permission for the fMRI studies ☐

4. I give my permission for my GP to be told of my involvement in this research ☐

Name of Patient Date Signature of patient

Name of Investigator Date Signature of investigator

1 copy for patient, 1 copy for research, 1 copy to be kept with hospital notes

Appendix 7 – Western Ontario Shoulder Instability Index

www.orthopaedicscores.com

Date of completion
May 19, 2012

The Western Ontario Shoulder Instability Index (WOSI)

Clinician's name (or ref) _____ Patient's name (or ref) _____

The following questions concern the symptoms you have experienced due to your shoulder problem. In all cases, please enter the amount of the symptom you have experienced in the last week. (please move the slider on the horizontal line.)

1. How much pain do you experience in your shoulder with overhead activities?

No pain — Extreme pain

2. How much aching or throbbing do you experience in your shoulder?

No aching/throbbing — Extreme aching/throbbing

3. How much weakness or lack of strength do you experience in your shoulder?

No weakness — Extreme weakness

4. How much fatigue or lack of stamina do you experience in your shoulder?

No fatigue — Extreme fatigue

5. How much clicking, cracking or snapping do you experience in your shoulder?

No clicking — Extreme clicking

6. How much stiffness do you experience in your shoulder?

No stiffness — Extreme stiffness

7. How much discomfort do you experience in your neck muscles as a result of your shoulder?

No discomfort — Extreme discomfort

8. How much feeling of instability or looseness do you experience in your shoulder?

No instability — Extreme instability

9. How much do your compensate for your shoulder with other muscles?

12. How much has your shoulder affected your ability to perform the specific skills required for your sport or work? (If your shoulder affects both sports and work, consider the area that is most affected.)

Not affected — Extremely affected

13. How much do you feel the need to protect your arm during activities?

Not at all — Extreme

14. How much difficulty do you experience lifting heavy objects below shoulder level

No difficulty — Extreme difficulty

15. How much fear do you have of falling on your shoulder?

No fear — Extreme fear

16. How much difficulty do you experience maintaining your desired level of fitness

No difficulty — Extreme difficulty

17. How much difficulty do you have "roughhousing" or "horsing around" with family or friends

No difficulty — Extreme difficulty

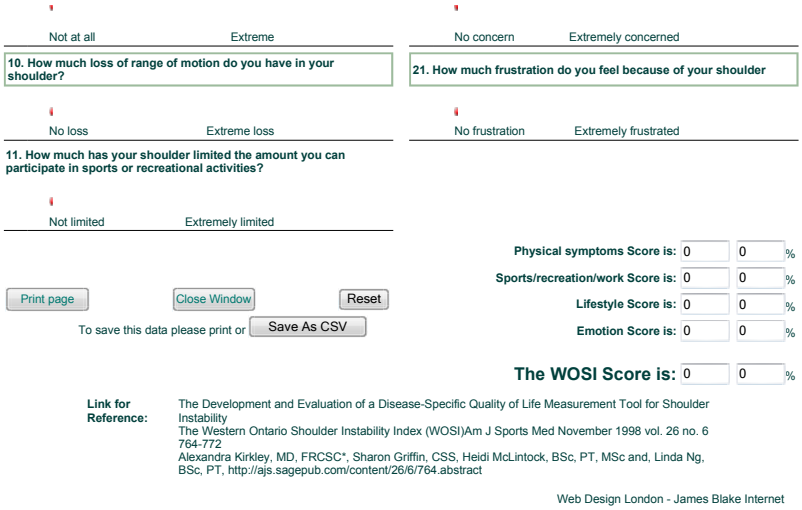
18. How much difficulty do you have sleeping because of your shoulder

No difficulty — Extreme difficulty

19. How conscious are you of your shoulder

Not conscious — Extremely conscious

20. How concerned are you about your shoulder becoming worse



Not at all Extreme

10. How much loss of range of motion do you have in your shoulder?

No loss Extreme loss

11. How much has your shoulder limited the amount you can participate in sports or recreational activities?

Not limited Extremely limited

No concern Extremely concerned

21. How much frustration do you feel because of your shoulder

No frustration Extremely frustrated

Print page Close Window Reset

To save this data please print or Save As CSV

Physical symptoms Score is:	0	0 %
Sports/recreation/work Score is:	0	0 %
Lifestyle Score is:	0	0 %
Emotion Score is:	0	0 %
The WOSI Score is:	0	0 %

Link for Reference: The Development and Evaluation of a Disease-Specific Quality of Life Measurement Tool for Shoulder Instability
The Western Ontario Shoulder Instability Index (WOSI)Am J Sports Med November 1998 vol. 26 no. 6 764-772
Alexandra Kirkley, MD, FRCSC*, Sharon Griffin, CSS, Heidi McLintock, BSc, PT, MSc and, Linda Ng, BSc, PT, http://ajs.sagepub.com/content/26/6/764.abstract

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Appendix 8 – Oxford Shoulder Instability Score

Oxford Shoulder Instability Score (OSIS)

English version for the United Kingdom

Prior to completing the Questionnaire please complete the following:-

Today's Date:

D	D	M	M	2	0	Y	Y

On which side of your body is the affected joint, **for which you are receiving treatment**.

Left ❑

Right ❑

Both ❑

If you said 'both', please complete the first questionnaire thinking about the right side. A second questionnaire, for the left side, will follow.

PROBLEMS WITH YOUR SHOULDER

Nb. not suitable for post-operative patients until 6 months

Tick (✓) one box for every question.

1. During the last 6 months...

How many times has your shoulder slipped out of joint (or dislocated)?

Not at all in 6 months	1 or 2 times in 6 months	1 or 2 times per month	1 or 2 times per week	More often than 1 or 2 times/week
☐	☐	☐	☐	☐

2. During the last 3 months...

Have you had any trouble (or worry) with putting on a T-shirt or pullover because of your shoulder?

No trouble/no worries	Slight trouble or worry	Moderate trouble or worry	Extreme difficulty	Impossible to do
☐	☐	☐	☐	☐

3. During the last 3 months...

How would you describe the **worst** pain you have had from your shoulder?

None	Mild ache	Moderate	Severe	Unbearable
☐	☐	☐	☐	☐

4. During the last 3 months...

How much has the problem with your shoulder interfered with your usual work? (including school or college work, or housework)

Not at all	A little bit	Moderately	Greatly	Totally
☐	☐	☐	☐	☐

5. During the last 3 months...

Have you avoided any activities due to worry about your shoulder – feared that it might slip out of joint?

No, not at all	Very occasionally	Some days	Most days or more than one activity	Every day or many activities
☐	☐	☐	☐	☐

6. During the last 3 months...

Has the problem with your shoulder prevented you from doing things that are important to you?

No, not at all	Very occasionally	Some days	Most days or more than one activity	Every day or many activities
☐	☐	☐	☐	☐

7. During the last 3 months...

How much has the problem with your shoulder interfered with your social life? (including sexual activity – if applicable)

Not at all	Occasionally	Some days	Most days	Every day
☐	☐	☐	☐	☐

8. During the last 4 weeks...

How much has the problem with your shoulder interfered with your sporting activities or hobbies?

Not at all	A little/ occasionally	Some of the time	Most of the time	All of the time
☐	☐	☐	☐	☐

9. During the last 4 weeks...

How often has your shoulder been 'on your mind' – how often have you thought about it?

Never, or only if someone asks	Occasionally	Some days	Most days	Every day
☐	☐	☐	☐	☐

10. During the last 4 weeks...

How much has the problem with your shoulder interfered with your ability – or willingness – to lift heavy objects?

Not at all	Occasionally	Some days	Most days	Every day
☐	☐	☐	☐	☐

11. During the last 4 weeks...

How would you describe the pain you usually had from your shoulder?

None	Very mild	Mild	Moderate	Severe
☐	☐	☐	☐	☐

12. During the last 4 weeks...

Have you avoided lying in certain positions, in bed at night, because of your shoulder?

No nights	Only 1 or 2 nights	Some nights	Most nights	Every night
☐	☐	☐	☐	☐

Finally, please check back that you have answered each question.
Thank you very much.

Appendix 9 – The Beck's Depression Inventory

Beck's Depression Inventory

This depression inventory can be self-scored. The scoring scale is at the end of the questionnaire.

1.
- 0 I do not feel sad.
- 1 I feel sad
- 2 I am sad all the time and I can't snap out of it.
- 3 I am so sad and unhappy that I can't stand it.

2.
- 0 I am not particularly discouraged about the future.
- 1 I feel discouraged about the future.
- 2 I feel I have nothing to look forward to.
- 3 I feel the future is hopeless and that things cannot improve.

3.
- 0 I do not feel like a failure.
- 1 I feel I have failed more than the average person.
- 2 As I look back on my life, all I can see is a lot of failures.
- 3 I feel I am a complete failure as a person.

4.
- 0 I get as much satisfaction out of things as I used to.
- 1 I don't enjoy things the way I used to.
- 2 I don't get real satisfaction out of anything anymore.
- 3 I am dissatisfied or bored with everything.

5.
- 0 I don't feel particularly guilty
- 1 I feel guilty a good part of the time.
- 2 I feel quite guilty most of the time.
- 3 I feel guilty all of the time.

6.
- 0 I don't feel I am being punished.
- 1 I feel I may be punished.
- 2 I expect to be punished.
- 3 I feel I am being punished.

7.
- 0 I don't feel disappointed in myself.
- 1 I am disappointed in myself.
- 2 I am disgusted with myself.
- 3 I hate myself.

8.
- 0 I don't feel I am any worse than anybody else.
- 1 I am critical of myself for my weaknesses or mistakes.
- 2 I blame myself all the time for my faults.
- 3 I blame myself for everything bad that happens.

9.
- 0 I don't have any thoughts of killing myself.
- 1 I have thoughts of killing myself, but I would not carry them out.
- 2 I would like to kill myself.
- 3 I would kill myself if I had the chance.

10.
- 0 I don't cry any more than usual.
- 1 I cry more now than I used to.
- 2 I cry all the time now.
- 3 I used to be able to cry, but now I can't cry even though I want to.

11.

0 I am no more irritated by things than I ever was.
1 I am slightly more irritated now than usual.
2 I am quite annoyed or irritated a good deal of the time.
3 I feel irritated all the time.

12.

0 I have not lost interest in other people.
1 I am less interested in other people than I used to be.
2 I have lost most of my interest in other people.
3 I have lost all of my interest in other people.

13.

0 I make decisions about as well as I ever could.
1 I put off making decisions more than I used to.
2 I have greater difficulty in making decisions more than I used to.
3 I can't make decisions at all anymore.

14.

0 I don't feel that I look any worse than I used to.
1 I am worried that I am looking old or unattractive.
2 I feel there are permanent changes in my appearance that make me look unattractive
3 I believe that I look ugly.

15.

0 I can work about as well as before.
1 It takes an extra effort to get started at doing something.
2 I have to push myself very hard to do anything.
3 I can't do any work at all.

16.

0 I can sleep as well as usual.
1 I don't sleep as well as I used to.
2 I wake up 1-2 hours earlier than usual and find it hard to get back to sleep.
3 I wake up several hours earlier than I used to and cannot get back to sleep.

17.

0 I don't get more tired than usual.
1 I get tired more easily than I used to.
2 I get tired from doing almost anything.
3 I am too tired to do anything.

18.

0 My appetite is no worse than usual.
1 My appetite is not as good as it used to be.
2 My appetite is much worse now.
3 I have no appetite at all anymore.

19.

0 I haven't lost much weight, if any, lately.
1 I have lost more than five pounds.
2 I have lost more than ten pounds.
3 I have lost more than fifteen pounds.

20.

0 I am no more worried about my health than usual.
1 I am worried about physical problems like aches, pains, upset stomach, or constipation.
2 I am very worried about physical problems and it's hard to think of much else.
3 I am so worried about my physical problems that I cannot think of anything else.

21.

0 I have not noticed any recent change in my interest in sex.
1 I am less interested in sex than I used to be.
2 I have almost no interest in sex.
3 I have lost interest in sex completely.

INTERPRETING THE BECK DEPRESSION INVENTORY

Now that you have completed the questionnaire, add up the score for each of the twenty-one questions by counting the number to the right of each question you marked. The highest possible total for the whole test would be sixty-three. This would mean you circled number three on all twenty-one questions. Since the lowest possible score for each question is zero, the lowest possible score for the test would be zero. This would mean you circles zero on each question. You can evaluate your depression according to the Table below.

Total Score_____Levels of Depression

1-10_____These ups and downs are considered normal
11-16_____ Mild mood disturbance
17-20_____Borderline clinical depression
21-30_____Moderate depression
31-40_____Severe depression
over 40_____Extreme depression

A PERSISTENT SCORE OF 17 OR ABOVE INDICATES THAT YOU MAY NEED MEDICAL TREATMENT. IF YOU HAVE ANY CARDIAC CONCERNS, PLEASE CONTACT CARDIOVASCULAR INTERVENTIONS, P.A. at 407-894-4880